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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

III.—ANIMAL INVESTIGATIONS.

(a)—BEHAVIORAL, ELECTRICAL AND ANATOMICAL STUDIES OF ABNORMAL EARS.*†

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Introduction: Two methods are available for measuring hearing acuity in animals. One is the conditioning or training method, along the lines developed by Pavlov (for example, see Andreyev, 1936). To test an animal's hearing acuity by this procedure, one must condition the animal to respond to tone stimuli of threshold value with some definite activity, such as opening a food box. This procedure is really not very different from clinical hearing tests in man, in which the subject is required, for example, to press a key when he hears a sound. The second method is the electrical, described by Wever and Bray (1930). Both methods have been applied separately to normal animals and to animals with defective hearing. A complete understanding of the relation of deaf-

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ness to abnormalities of the hearing mechanism requires a combination of the two methods on the same animal, followed by microscopic studies of the anatomical abnormalities underlying the loss of function. We have been engaged upon such a study for the past year, and, although experiments are still in progress, the results so far obtained are sufficient to warrant presentation.

METHOD.

One of us (Dworkin) trained a number of cats to open a food box whenever they heard a tone of any pitch. One cochlea of each animal was then destroyed aseptically, and at a second operation a partial lesion of the other cochlea or of the ossicles was produced by drill or cautery. Training was then resumed and the threshold of response to frequencies 100, 400, 1,000, 3,000, 5,000, 10,000 and 16,000 was determined. The tests spread over a period of three to six months. Four to 15 determinations, which usually agreed with one another within $\pm$ 7 dcb., were obtained at each frequency, and the average value was compared with the thresholds of normal animals under identical conditions. The losses plotted in the usual way constitute a true audiogram for the partially damaged ear. On account of the time required for the training and testing it was deemed advisable to limit the tests to the seven frequencies listed above. During the test period due attention was given to the cleanliness of the external auditory canals.

Within a week of the conclusion of the conditioned-reflex studies the animals were tested electrically by Davis.* The apparatus and methods have already been described (Dworkin, 1934, 1935) and consist in general in determining the intensity of sound necessary at various frequencies to produce a just-detectable electrical response recorded from the round window. The loss in sensitivity was estimated by comparing the thresholds of response with the average thresholds of six normal cats determined with the same apparatus under identical conditions.

The physiological tests were carried out under avertin or dial and urethane anesthesia. The scope of the earlier tests

*We acknowledge our gratitude for assistance in the conduct of these tests to Dr. E. H. Kemp and Dr. H. C. Wiggers.

was unfortunately limited by a defective voice coil in the loud-speaker used to produce the stimulating sound, which limited the intensity of tones below 400 c.p.s., which could be generated without introducing harmonics which seriously complicated the waveform. This defect was detected and remedied after the first four animals had been studied. We were also limited in testing the hearing loss at frequencies above 8,000 c.p.s. Our stimulating apparatus was incapable at these frequencies of delivering sounds of sufficient intensity to produce a threshold response unless the sensitivity of the animal was normal or nearly so. Even the normal thresholds are less certain and satisfactory than for lower

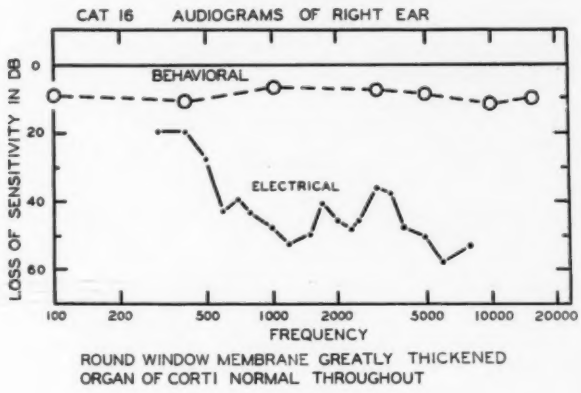

Fig. 1.

tones, since the maximal electrical response is very small at these high frequencies and a response much less than maximal may escape detection if conditions for electrical recordings are not ideal.

The ears were fixed, sectioned and examined microscopically by Lurie. Utilizing our previous experience with guinea pigs (Stevens, Davis and Lurie, 1935), Lurie, in ignorance of the results of the behavioral and electrical tests, estimated the degree of impairment of hearing for various tones on the basis of the position and extent of the pathological lesions observed under the microscope. Presence or absence of the nuclei of the hair cells of the organ of Corti served as the chief criterion of judgment.

RESULTS.

Both ears of each animal were tested and examined microscopically to control the completeness of the destruction of one ear at the original operation. In all cases this control was satisfactory, although in two cases a slight electrical response at intensities 80 dcb. or more above threshold was detected in the most sensitive part of the audible scale. These vestigial responses correlated satisfactorily with the histological findings, which showed remnants of damaged organs of Corti; but in no case was the sensitivity of the "destroyed" ear sufficient to have contributed to the hearing of the animal as determined by the conditioned reflex to sounds.

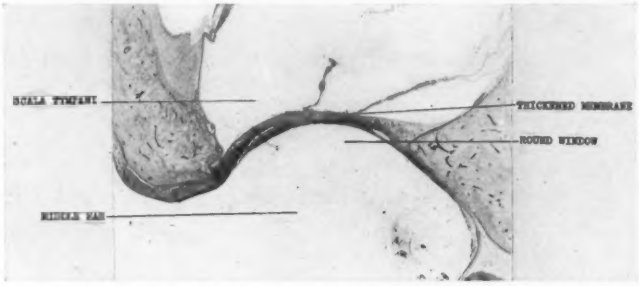

Fig. 2. Photomicrograph, 50 X. Round window of cat, showing marked thickening of the membrane.

Fig. 1 shows the electrical audiogram of an animal which by its conditioned responses showed quite normal acuity of hearing. Electrically there is a loss of sensitivity of 20 dcb. at 300 and 400 c.p.s. For higher frequencies the sensitivity is more and more depressed. The maximum loss measured was 58 dcb. at 6,000 c.p.s. On microscopic examination the middle and also the inner ear appeared normal except for some fibrous tissue filling the head of the stapes and a marked thickening of the round-window membrane (see Fig. 2). The attempt to produce partial destruction of the cochlea had been quite unsuccessful. The prediction from anatomical evidence was for normal hearing, which agrees with the behavioral test. The marked deviation of the electrical test is explained by the great thickening of the round-window membrane. It was

noted at the time of test that the maximal electrical response was only 20 μv. instead of the usual 1,000 μv. Evidently the thickened round-window membrane prevented the electrical disturbance generated in the cochlea from reaching the electrodes at normal intensity. This led to an apparent depression of sensitivity. The greater depression of high tones than low is partly due to a progressive failure of response during the electrical test. Readings were taken in order from low to high and repetition of frequencies between 350 and 1,000 c.p.s. showed a rise of 20 dcb. in threshold, and the maximal response at 1,000 c.p.s. had fallen to 10 μv. Such progressive failure is exceptional during our tests.

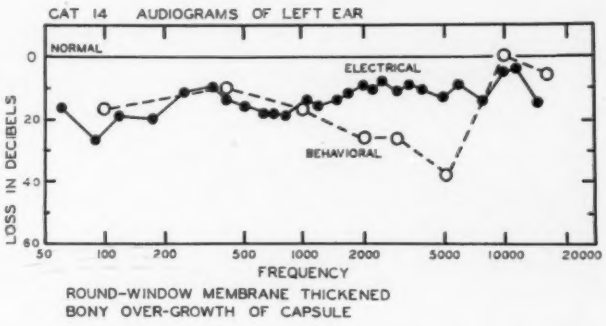

Fig. 3.

Fig. 3 shows the electrical and behavioral audiograms obtained from another cat. Both agree in showing practically normal hearing at 10,000 and 16,000, and a slight depression at 1,000 and below. The depression for low tones never exceeds 20 dcb. The first behavioral test showed normal hearing at 2,000, 3,000 and 5,000 also, but later tests showed consistent elevations of threshold averaging 26, 26 and 38 dcb., respectively.

Anatomically, the middle and inner ears were normal except for: *1.* moderate thickening of the round-window membrane; *2.* a new growth of bone on the capsule near the round window, extending to the spiral ligament; and *3.* possible degenerative changes in a few external hair cells in the first

half of the basal turn. The moderate thickening of the round-window membrane is probably enough to account for the slight depression of the electrical audiogram. There is no abnormality to account for the loss of sensitivity at 2,000, 3,000 and 5,000 c.p.s. in the conditioned reflex tests. The ganglion cells and nerve appeared perfectly normal. Lurie's prediction was for normal hearing or a slight loss of not more than 20 dcb. at most. This prediction is confirmed by the results of the electrical tests.

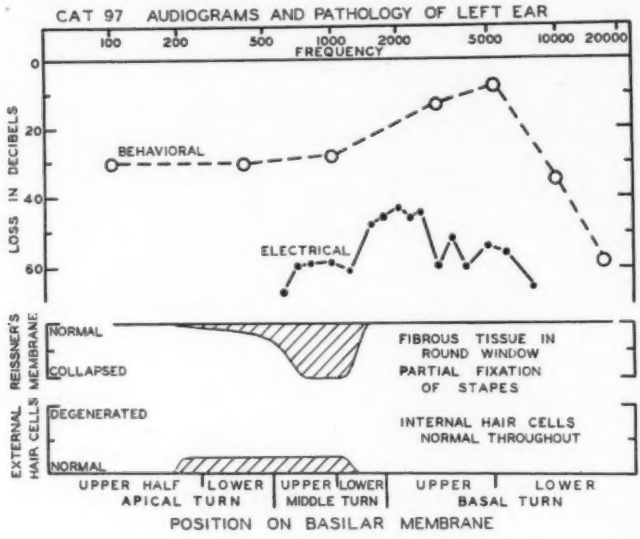

Fig. 4.

The electrical and behavioral audiograms from still another cat are shown in Fig. 4. The behavioral tests showed nearly normal hearing at 3,000 and 5,000, and depressions of 27 dcb. or more at the other frequencies which were tested. The electrical audiogram runs approximately parallel, but the determinations below 600 are questionable, since only harmonics without the fundamental appeared at threshold. The average depression is about 60 dcb. Sensitivity is a little better than this between 1,500 and 2,500, but no behavioral

tests were made at these frequencies. The histological examination showed nothing whatever to correspond to the high-tone loss, as the organ of Corti appeared normal in the basal turn and the lower half of the middle turn. In the apical and the upper half of the middle turn, however, there were definite degenerative changes in the external hair cells, and Reissner's membrane was either collapsed or actually down in contact with the organ of Corti. Saccule, utricle and semicircular canals were also collapsed but showed normal neuroepithelium. On the basis of these findings, Lurie predicted a partial loss of hearing below 1,200 and probably harmonics in the electrical response below 2,000 because of the collapse of Reissner's membrane (cf. Davis, Derbyshire, Kemp, Lurie and Upton, 1935). The electrical test fulfilled the latter expectation, in that strong harmonics were present below 1,500 and completely dominated the picture below 500. In fact, the rather moderate and equal loss for the three lowest frequencies by the behavioral test suggests that the animal may have been responding to the harmonics rather than to the fundamental tone.

The greater average loss by electrical test as compared to the reflex test in this case is reasonably explained by the condition of the round window, which was filled with fibrous tissue. The stapes was also imbedded in fibrous tissue and partially fixed. The latter abnormality would, of course, contribute to the loss of low-tone sensitivity. If we are justified in assuming a loss of 40 or 50 dcb. in the sensitivity of the electrical pickup because of shunting by the fibrous tissue in the round window, the three sets of observations show satisfactory agreement.

It is worth noting that in the opposite ear of this same animal whose cochlea had been completely destroyed, no electrical activity could be detected. The macula of the saccule and its nerve both appeared normal, however. This indicates that the saccule does not contribute to the electrical response, which we have ascribed to the cochlea.

Another animal in which both ears had been severely damaged responded on behavioral test to tones up to and including 1,500 c.p.s., but to no higher frequency. The hearing loss lay uniformly between 76 and 90 dcb. The responses below 1,000 c.p.s. must be discounted as due to tremor at

these high intensities and not to hearing (Dworkin, Hutchison and Katzman, 1936), but Dworkin characterized the responses at 1,200 and 1,500 c.p.s. as "probably true response." The point at 1,000 c.p.s. is questionable.

Electrical test of the right ear showed no response, while the left ear showed slight response between 1,200 and 2,250, with loss of sensitivity between 85 and 95 dcb. Histological examination showed some internal hair cells present in the lower half of the middle and the upper half of the basal turns, but none elsewhere. There was partial degeneration of the cochlear ganglion throughout. The saccule was col-

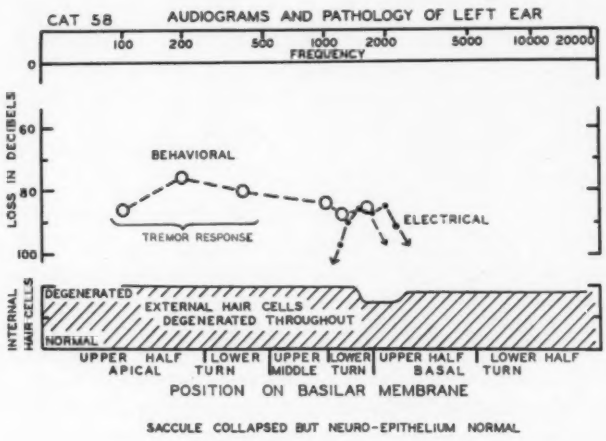

Fig. 5.

lapsed, but its neuroepithelium was normal. These findings led Lurie to predict "some slight response from 1,500 to 2,500, and possibly upward." The three sets of findings coincide closely (see Fig. 5) if allowance is made for the normal rise in threshold for frequencies over 2,500, which would preclude detection of response at the intensities of stimulation available if the hearing-loss were as great as 80 dcb.

A fifth animal was found by Dworkin to be very much depressed at all frequencies. This may have been due in part to the large amount of wax and the inflamed condition of the left external canal which was found when the elec-

trical tests were made. The right ear had been completely destroyed. By electrical test the animal showed a "hearing loss" in the left ear of approximately 44 dcb. at all frequencies at which satisfactory tests could be obtained. In addition to this, there is a sharp tonal gap centering at 850. The greatest loss of sensitivity here is 67 dcb. The sensitivity is slightly better between 3,000 and 4,000 than at other frequencies. The behavioral tests did not reveal the tonal gap at 850 c.p.s. The general depression of the behavioral threshold below that for electric response may depend upon

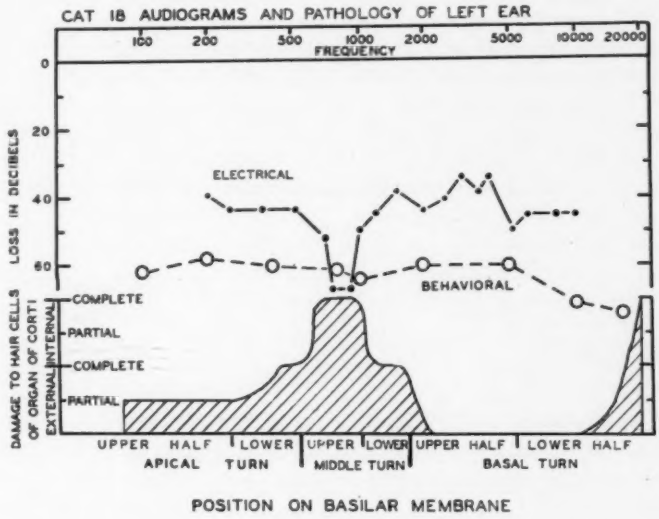

Fig. 6.

partial blockage of the external canal, which was relieved at the final operation.

Histological examination revealed a complex set of lesions. The eardrum was thickened and the middle ear filled with pus and exudate. The round-window membrane was moderately thickened. These conditions would lead us to expect at least a moderate loss of sensitivity at all frequencies. Saccule and utricle were normal. A drill hole entering the second turn of the cochlea had stimulated new bone formation, pro-

truding into the scala vestibuli and the spiral ligament of the scala media. The basilar membrane and Reissner's membrane were intact, but the organ of Corti had completely degenerated in this region. Movement of fluid past the lesion was not obstructed. On the basis of this local lesion, Lurie predicted severe loss of hearing from 750 to 1,500 c.p.s.—which is a very satisfactory check with the tonal island revealed electrically. The apical regions showed hair cells present, but partially degenerated, and some degeneration of the ganglion cells. In Fig. 6, the degree and location of abnormality of the hair cells of the organ of Corti are represented graphically. It is noteworthy that degeneration of the external hair cells extended much farther on both sides of the initial lesion than did degeneration of the internal hair cells. The basilar membrane in the figure is laid off along the scale of frequencies according to the map presented by Stevens, Davis and Lurie (1935) for the location of the various frequencies in the human cochlea. Unfortunately, limitations of the apparatus did not allow electrical tests at sufficiently high intensities to determine the threshold above 8,000, but the audiogram shows a zone of slightly greater sensitivity between 3,000 and 4,000, which is associated with a normal organ of Corti in the corresponding position.

DISCUSSION.

These cases taken as a whole show a fairly satisfactory correlation between the behavioral, the electrical and the histological examinations of the auditory mechanism. The correspondence between the histological findings and the electrical tests is particularly close. It should be noted that the predictions from the microscopic appearance as to the physiological performance were based primarily on the presence of the nuclei of the hair cells. Absence of external hair cells while the internal hair cells were present was taken to indicate an elevation of electrical threshold by some 30 dcb. The predictions as to the freqency at which changes in sensitivity would be found were based upon the map of the cochlea previously derived for guinea pigs and for humans (Stevens, Davis and Lurie, 1935). The close agreement with the actual findings seems to justify the conclusion that the position on the basilar membrane of maximal sensitivity for particular

frequencies follows the same distribution in the cat as in the guinea pig and human, and constitutes further confirmation of the "place" theory of pitch perception.

An important limitation of the electrical method for the study of the sensitivity of the inner ear is revealed by the apparent loss of sensitivity associated with thickening of the round-window membrane. As pointed out above, such loss of sensitivity is only apparent and is easily understood on the basis of purely physical hindrance of electrical current-flow. This factor must be carefully considered in the evaluation of tests by the electrical method.

One animal revealed a raised threshold for two particular tones when tested by its conditioned reflexes, although electrically and anatomically the organ of hearing appeared perfectly normal. This may indicate a limitation of the method of conditioned reflexes depending on idiosyncrasies of individual animals, or on the other hand, may reveal a case of actual partial deafness, depending upon some undetected lesion of the higher nervous pathways.

Within these limitations, however, we believe that the results obtained by the electrical method of testing fairly represent the true physiological performance of the ear, and that abnormalities of the threshold for electrical activity may be understood in detail in terms of anatomical abnormalities. We base this conclusion primarily on the correspondence between local damage to or survival of the organ of Corti and tonal gaps or islands. It is obvious to the skeptic, however, that the agreement is not perfect in all details, and we must consider also the observations of Bast and Eyster (1935), and particularly those of Ashcroft, Hallpike and Rawdon-Smith (1937). These investigators do not find any clear correlation between electrical activity and the condition of the organ of Corti, and ascribe the origin of the electrical phenomena to vibration of electrically polarized membranes in the inner ear. We are at present unable to explain all of their observations in terms of our view. They have measured the maximal electrical activity rather than the thresholds and this may prove to be the basis of the discrepancy. Experiments now in progress in which both threshold and maximal response are to be measured may clarify the situation. If Reissner's membrane or the basilar membrane are

actually polarized to a significant degree, their vibrations would indeed generate such potentials as are observed. We believe that potentials from this source are practically negligible, since we have failed to find electrical activity in ears without organ of Corti but with intact membranes, and a generalized polarization should not yield correlations such as we regularly find between abnormalities of the audiogram and local lesions. For the present we therefore adhere to our view that both actual hearing and the electrical activity of the cochlea depend upon the integrity of the organ of Corti.

The condition of the saccule showed no relation either to hearing or to the presence of electrical activity. Two animals which were totally deaf by behavioral test and showed no electrical activity and completely damaged organs of Corti in each ear had normal saccules on one or both sides, or showed at most a partial collapse of the saccule. A normal saccule did not enable the animal to hear. One other ear was electrically inactive but had a normal saccule, and in two other inactive ears the neuroepithelium was normal but the membranes collapsed. In these last three animals the opposite ear was partly functional and allowed the animal to respond on the behavioral test. All of this is strong evidence against participation by the saccule in the function of hearing.

SUMMARY.

Partial destruction of the organ of Corti was produced surgically in cats and the threshold of hearing was subsequently determined over a period of months by the method of conditioned reflexes. The sensitivity of the ears was tested at acute experiment by the electrical method, and the ears were finally studied microscopically.

Thickening of the round-window membrane (with intact organ of Corti) caused little or no depression of actual hearing but considerable loss of electrical response. This is probably due entirely to hindrance of electrical current flow. Otherwise the degree and extent of damage to the organ of Corti, revealed by absence of nuclei of the hair cells, corresponded to general or local depressions of the electrical audiogram. The frequency range and the degree of loss of sensitivity were estimated with considerable accuracy in all cases on

the basis of the anatomical lesions. The location of particular frequencies on the basilar membrane of the cat is the same as in guinea pig and human.

The correspondence between the behavioral tests and the anatomical and electrical findings was good, but not perfect.

The saccule does not contribute to hearing or to the electrical activity of the inner ear.

REFERENCES.

ANDREYEV, L.: Physiology of the Acoustic Analyzer of the Dog Studied by Means of the Method of Conditioned Reflexes. *Bull. de Biol. et de Méd. Expér.*, 1:183-184, 1936.

ASHCROFT, D. W.; HALLPIKE, C. S., and RAWDON-SMITH, A. F.: On the Changes in Histological Structure and Electrical Response of the Cochlea of the Cat Following Section of the VIIIth Nerve. *Proc. Roy. Soc. London*, Ser. B, 122:186-197, 1937.

BAST, T. H., and EYSTER, J. A. E.: The Function of the Apical Turns of the Cochlea and the Symptoms of a Lesion in This Location. Discussion from the Point of View of Animal Experimentation. *Trans. Amer. Otol. Soc.*, 1935.

DAVIS, H.; DERBYSHIRE, A. J.; KEMP, E. H.; LURIE, M. H., and UPTON, M.: Functional and Histological Changes in the Cochlea of the Guinea Pig Resulting from Prolonged Stimulation. *Jour. Gen. Psychol.*, 12:251-278, 1935.

DWORKIN, S.: Hearing Tests in Normal and Operated Dogs and Cats. *Trans. Amer. Otol. Soc.*, 1934. Pitch and Intensity Discrimination by Cats. *Amer. Jour. Physiol.*, 112:1-4, 1935.

DWORKIN, S.; HUTCHISON, G. A., and KATZMAN, J.: Tactile and Tremor Perception. (In Symposium Before the Amer. Otol. Soc., May 28, 1936, Detroit.) *Ann. Otol., Rhinol. and Laryngol.*, 45:840-844, 1936.

STEVENS, S. S.; DAVIS, H., and LURIE, M. H.: The Localization of Pitch Perception on the Basilar Membrane. *Jour. Gen. Psychol.*, 13:297-315, 1935.

WEVER, E. G., and BRAY, C. W.: The Nature of Acoustic Response: the Relation Between Sound Frequency and Frequency of Impulses in the Auditory Nerve. *Jour. Exper. Psychol.*, 13:373-387, 1930.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

III.—ANIMAL INVESTIGATIONS.

(b)—TOPOGRAPHY OF THE ACOUSTIC SYSTEM IN COCHLEA AND MEDIAL GENICULATE BODIES.*†

E. A. CULLER, Ph.D., Urbana, Ill.

Our immediate program at the Animal Hearing Laboratory, University of Illinois, consists in mapping the resonant foci of the cochlea, the acoustic pathways of the medial geniculate bodies and the sensory termini of the auditory cortex.

Two years ago, at the Toronto meeting of this Society, a map of the guinea-pig cochlea was published from our Laboratory, based upon a careful survey of cochlear potentials generated by air-borne frequencies ranging from 60 to 7,000 cycles. The methodology employed in deriving this map was fully explained at the time and in the printed article (*Ann. Otol., Rhinol. and Laryngol.*, 44:807, 1935) and need not be repeated now. This survey differed from all similar efforts in that the cochlea, instead of being drugged, punctured or cauterized, was in every respect normal, a sharp needle-electrode being applied to the external surface of the intact cochlea. This map revealed that each audible frequency has its own focus of response within the cochlea and that the site of a given focal point can be determined within narrow limits. Such a chart, while interesting, is not wholly conclusive until corroborated by hearing-tests after localized lesions; that is, one cannot be certain of the *auditory* significance of these potential-foci until hearing tests have been made. Such tests encounter numerous difficulties in the guinea-pig. This curious beast has a marvelous ear, but its brain leaves much

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to be desired; training it to react reliably to a tone is, therefore, a slow and painstaking business. The delicate and involved respiratory mechanism is extremely susceptible to operative invasions of the middle ear, so that mortality is high. Despite all this, tests have now been completed for seven animals at one critical frequency, 200 cycles, which appears on our chart at mid-ventral aspect of the first turn below apex. Normal thresholds of acuity are first located for a single ear, the other ear having previously been destroyed. The cochlear wall is then punctured, near point 200 of the map, with a surgical needle; the tiny perforation at first oozes lymph but soon (within several days, as was also reported by Kobrak for rabbits) closes over, thus restoring the cochlea into a closed hydrodynamic system for test-purposes. We consider this of some importance since it is accepted that the hydrodynamic behavior of a closed system is appreciably altered when even a small vent is opened at one point. Figures from three representative animals (Table I) may be cited (hearing-losses at various frequencies after lesion of cochlear potential-focus for 200 cycles).

TABLE I.

HEARING LOSSES AFTER LESION AT COCHLEAR FOCUS FOR 200 CYCLES.

	125	150	200	250	300	400	500	700	1,000 cycles
1.	17	16	*25*	22	15	—	14	9	4 decibels
2.	34	33	33	*36*	35	—	30	25	26 decibels
3.	29	31	35	*39*	38	34	35	33	30 decibels

Here, as in all seven of the pigs thus far tested, we see that maximal hearing-loss occurs at 200 or 250 cycles, which agrees well though not perfectly with the charted frequency; the impairment furthermore extends over a wide range of frequencies, though it consistently scrapes bottom around 200-250. From this evidence the following conclusions are tentatively inferred:

a. The resonant principle, being validated by the accordant testimony of both cochlear response and hearing-tests, seems to be proved beyond any reasonable doubt.

b. This material likewise provides evidence of direct agreement betwen cochlear response and hearing.

The medial geniculate body being the final way-station from ear to cortex, its importance for hearing need not be argued. By means now available, it is possible both to evaluate the hearing-losses consequent upon destruction of any given area and also to measure the potentials generated by nerve-impulses on their way through this centre. Some results by the former of these methods are now ready to report.

Auditory limens for pure tones are obtained in cats by the conditioned-response technique on the seven octaves from 125 to 8,000 cycles. By means of the Horsley-Clarke stereotaxic instrument (carefully calibrated for cats of this size),

TABLE II.

LESIONS IN MEDIAL GENICULATE BODIES

Cat #49 Symmetrical lesions in m.g.b.

Freq.	125	250	500	1000	2000	4000	8000	cycles
pre.	72	77	78	86	82	78	74	decibels
post.	70	78	76	85	78	62	74	"
diff.	-2	+1	-2	-1	-4	-16	0	"

Cat #42 Left m.g.b. partially destroyed.

Freq.	125	1000	8000	cycles
pre.	61	80	60	decibels
post.	58	71	56	"
diff.	-3	-9	-4	"

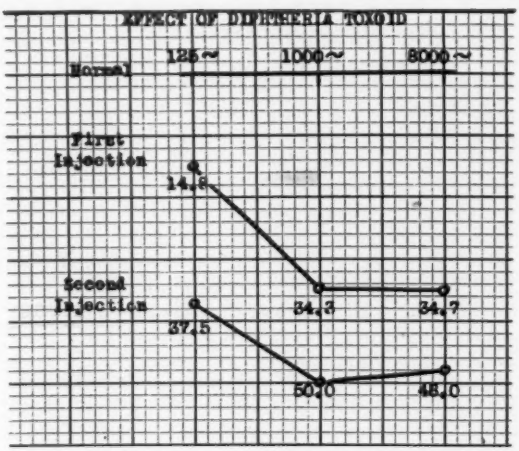
Cat #40 Left m.g.b. completely destroyed.

Freq.	125	1000	8000	cycles
pre.	64	80	63	decibels
post.	53	70	54	"
diff.	-11	-10	-9	"

localized electrolytic lesions are made bilaterally (symmetrically) in the medial geniculate bodies. Here again special procedures must be devised. First, the ear-plugs by which the instrument is aligned to the interaural axis must be short enough to obviate rupture of the tympanum and yet long enough to insure firm anchorage in the bony meatus. Secondly, asepsis is unusually difficult since the concha must be deeply incised between tragus and antitragus for admittance of the plugs and must then heal perfectly to insure reliable postoperative tests. A unipolar electrode of tungsten-wire, insulated with enamel save for the tip which penetrates some part of the geniculates, is attached to the negative terminal

of a B-battery; the circuit is completed via the temporal muscle, which is overlaid with a cotton plug soaked in saline. After a period of one or two days, the cats are again tested on the same frequencies to locate the nature and degree of hearing-loss.

Table II summarizes the figures I wish to present. *1.* In cat No. 40 complete destruction of one (left) medial geniculate body was attended by a loss of about 10 dcb. throughout the entire range of low, medial and high frequencies. *2.* Incomplete destruction in cat No. 42 of a single (left)



geniculate produced on the contrary a *differential* effect: liminal acuity was impaired at 1,000 cycles about the same as before (9 dcb.), whereas higher and lower frequencies were but little affected. *3.* After symmetrical lesions in both geniculates (No. 49), the audiogram was perhaps most striking of all: a sharp negative spike at 4,000, with no appreciable loss elsewhere.

The following inferences seem to be warranted: *a.* Complete destruction of a single geniculate causes a general subsidence at all frequencies comparable to the loss from destruction

of one hemicortex or one cochlea. *b*. Incomplete destruction of a single geniculate causes a restricted, instead of a general, subsidence. *c*. Localized bilateral lesions affect some frequencies far more than others, proving that the several frequencies traverse the geniculates by differential pathways. In other words, the topographical distribution of frequencies which begins in the cochlea persists at least as far as the geniculates.

Work on the auditory cortex, while well under way, does not yet permit any assured generalizations.

University of Illinois.

ADVISORY BOARD FOR MEDICAL SPECIALTIES.

The annual meeting of the Advisory Board for Medical Specialties, which is the coordinating board of the 12 certifying boards in the various specialties, the Association of Medical Colleges, the American Hospital Association, the Federation of State Medical Boards of the U. S. A., and the National Board of Medical Examiners was held at Atlantic City, N. J., on June 6, 1937.

The following officers were elected: Dr. Willard C. Rappleye, President, New York, N. Y.; Dr. W. P. Wherry, Vice-President, Omaha, Neb.; Dr. Paul Titus, Secretary-Treasurer, Pittsburgh, Pa. Executive Committee: Dr. W. B. Lancaster, Boston, Mass., and Dr. R. C. Buerki, Madison, Wis.

Dr. Louis B. Wilson, of Rochester, Minn., the retiring president of the board, was elected an emeritus member of the board.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

III.—ANIMAL INVESTIGATIONS.

(c)—ANIMAL EXPERIMENTS ON THE MECHANISM OF THE ACOUSTIC IRRITATION IN THE COCHLEA.*†

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Experiments† have been performed which aim to throw light upon the mechanism by which the nerve endings in Corti's organ are stimulated. The physiological stimulus of the cochlea consists in dynamic air pressure changes which are transformed into vibrations of the intralabyrinthine fluid. Since static pressure changes are easier to analyze, they were studied first. The displacement of the intralabyrinthine fluid due to static pressure changes in the outer acoustic canal and in the middle ear cavity was measured by the following method:

A tube was inserted into the outer acoustic meatus of a rabbit and made airtight. A second tube was fixed into the middle ear, and this opening was also firmly closed. Both tubes were connected with a syringe in which changes in pressure could be produced. A second connection was made with a manometer, by means of which the pressures were measured. The inner ear of the animal was opened and a tiny glass capillary was inserted. By an optical system the meniscus was visualized on a screen; movements of the meniscus due to changes in pressure in either the middle or the outer ear could be observed fairly accurately (see Fig. 1).

The chart in Fig. 2 records the readings for the displacements of the labyrinthine fluid due to various positive and negative pressures in both the outer and the middle ear.

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*Read as part of the Symposium, "The Neural Mechanism of Hearing," at the Seventieth Annual Meeting of the American Otological Society, Long Beach, N. Y., May 27, 1937.

†The experiments were carried out in collaboration with Dr. J. R. Lindsay and Dr. H. B. Perlman. I also wish to thank the Sonotone Co. for furnishing one of their instruments.

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The result was striking, in that changes in pressure in the middle ear produced a far greater movement of labyrinthine fluid than identical changes in the outer ear. From the quantitative finding one may assume that minor pressure changes in the middle ear, as occurring during swallowing or yawning, are effective on the inner ear. However, functional tests reveal that the function of the cochlea is not impaired as long as these static pressure changes are relatively small.

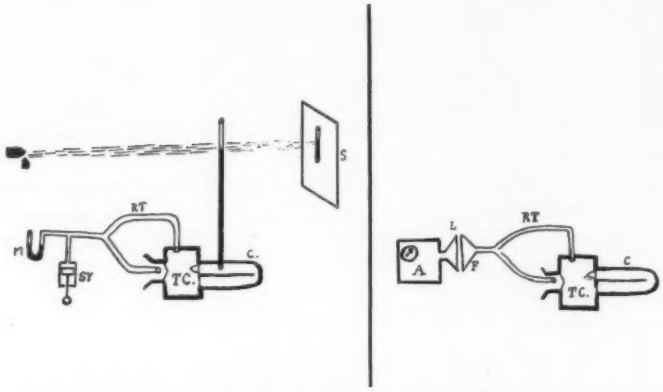

Fig. 1. Diagram illustrating the method. The left half of the figure demonstrates the arrangement for static experiments. The right side shows the experimental procedure for acoustic stimulation. The static pressure change is produced in the syringe (SY), measured in the manometer (M) and conducted by rubber tubes (RT), either into the outer ear or into the tympanic cavity (TC). The meniscus of the capillary inserted into the cochlea (C) is visualized on the screen (S). In the dynamic experiments, oscillations of acoustic frequency are produced by the audiometer (A). A glass funnel (F) attached to the loud-speaker (L) leads the sound energy either into the outer ear or into the middle ear.

The experiments on dynamic (acoustic) pressure changes were carried out in a similar way (see Fig. 1).

Rabbits were used for the experiments. Rubber tubes were inserted into the outer acoustic canal and in the middle ear as in the static experiments. The tubes formed a Y leading to a glass funnel which was placed in front of a loud-speaker, thus collecting the sound energy from the sound producer. By clamping the tubes the application of the sound could be interrupted entirely or confined to the outer acoustic meatus or to the middle ear. The effect on the cochlea of the animal

was determined by the acoustic reflex contractions of the tensor tympani muscle. The reflex contractions can be used quantitatively as indicator of cochlear stimulation, both by recording or observation. In the majority of our experiments the threshold of contraction was read for various frequencies and plotted in curves.

The solid line in Fig. 3 shows the tensor curve for the sound conducted into the outer acoustic canal. As the curve shows, the threshold is raised considerably by application into the middle ear (broken line). In other words, the irritation

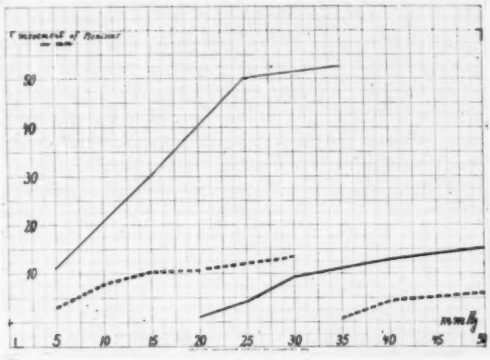

Fig. 2. Chart showing the displacement of the labyrinthine fluid due to changes in pressure in the outer and middle ear of the rabbit. The abscissae represent the changes in pressure (mm. Hg.); the ordinates indicate the movements of the meniscus of the capillary as read on the screen. The dotted line at the right represents positive pressures in the outer acoustic canal; the left dotted line indicates positive pressures in the tympanic cavity. The two solid lines represent negative pressures, the steeper one indicating pressure changes in the middle ear. Changes in pressure in the middle ear produce greater displacements of the intralabyrinthine fluid than equivalent changes in pressure in the outer acoustic meatus.

of the nerve endings in Corti's organ is definitely greater when the sound is conducted directly into the middle ear. This is similar to the effect of changes in static pressure, as shown in Fig. 2.

The role of the round window was investigated in experiments in which the niche of the round window was plugged by means of a small pledget of wet cotton. The result is seen in Fig. 4. By comparing Charts 3 and 4, one can see that the "hearing gain" by application to the middle ear was

diminished after the plugging, while, on the other hand, the hearing by way of the outer ear was practically unchanged. Corroborating these results was a case in which Nature did

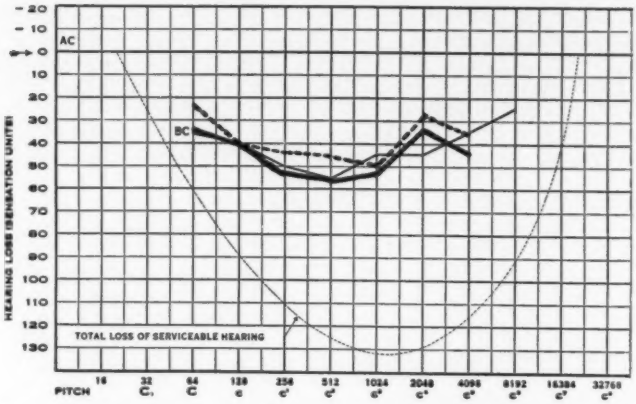

Fig. 3. Chart showing the thresholds of the tensor reflex of the rabbit for sound carried directly into the outer acoustic meatus (solid line) and for sound carried directly into the middle ear (dotted line).

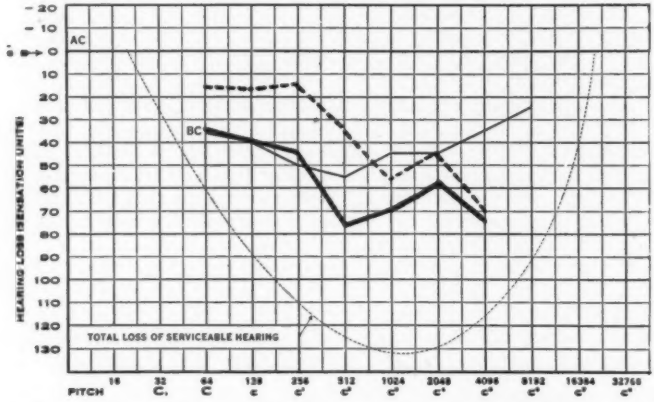

Fig. 4. Chart showing the thresholds of hearing after the niche of the round-window was plugged. The usual raise of threshold by application to the middle ear is diminished. The solid line indicates the threshold of the tensor reflex for sound carried directly into the outer ear; the dotted line, the threshold for sound carried directly into the middle ear.

an experiment similar to the artificial plugging. A rabbit which was tested failed to show the characteristic finding by application to the middle ear. This result was explained by

the histological examination of the middle ear (see Fig. 5). While the whole cavity of the bulla ossea was grossly free from pus, there was a residue of suppurative material in the region of the window. The niche was plugged rather tightly.

Another series of experiments demonstrated the effect of middle ear application in a pathological inner ear. In collaboration with Dr. F. H. Lewy, experiments have been carried out in which one coil of the cochlea has been destroyed. Fig. 6 shows an animal with a lesion in the basal coil. The

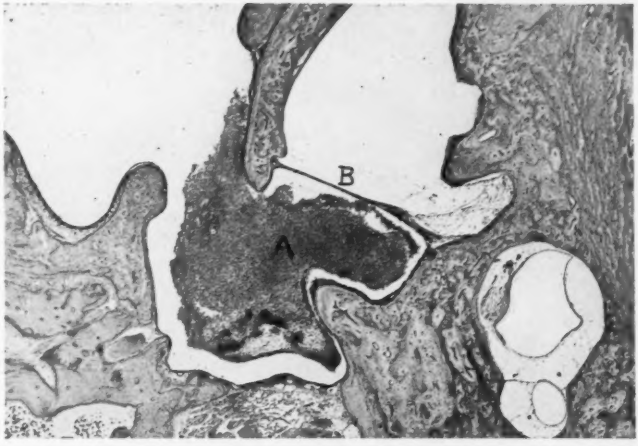

Fig. 5. Section of the middle ear, showing the niche of the round-window plugged by pus, accidentally observed in a rabbit which failed to show the typical "hearing gain" on application of sound to the middle ear. (A) indicates the accumulation of pus in the niche of the round-window, and (B), the membrane of the round-window and the scala tympani.

time between the operation and the sacrificing of the animal has been two weeks. The promontorial hole has closed. Corti's organ and Reissner's membrane are destroyed in the inferior coil. The adjacent gangli cells in Rosenthal's canal are degenerated. Following the cochlea lesion, a degeneration of fibres develops in the primary acoustic centres (see Fig. 7). The remaining function in this case of "artificial nerve deafness," determined immediately before sacrificing the animal, is given in the next curve (see Fig. 8, solid line). If one applies the sound into the middle ear it can be seen that the

pathological cochlea is stimulated stronger (see Fig. 8, broken line). Other animals had such severe cochlear lesions that there was no reflex function seen by outer ear application. Middle ear application, however, raised the threshold so that some cochlear function could be demonstrated. Besides in the rabbit, the phenomenon was studied in the cat. There is also evidence that the human cochlea shows the same property.

DISCUSSION.

The method of middle ear application seems to furnish a means of studying the nervous mechanism of hearing regard-

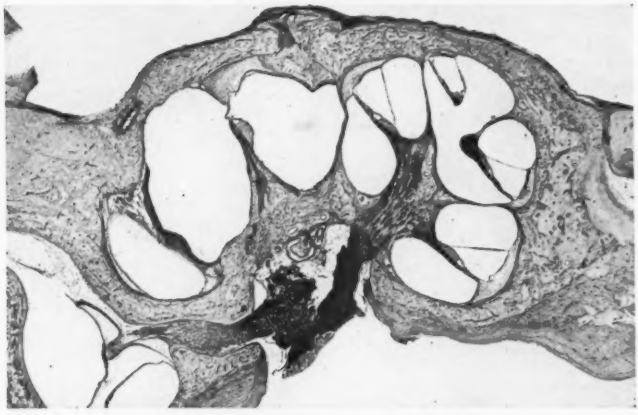

Fig. 6. Midmodiolar section of the cochlea of a rabbit with a lesion in the inferior coil. Superior and middle coil, as well as the modiolus, are intact. The bore in the promontorial wall extends into the scala vestibuli of the basilar turn. Connective tissue and newly-formed bone fill the bore and a part of the scala vestibuli. Reissner's membrane is destroyed. Remnants of it are to be seen attached to the spiral ligament. Corti's organ is destroyed. The nerve cells of the inferior spiral proved on microscopic examination to be degenerated.

ing an old physical question. It is considered uncertain which quality of the sound wave is the stimulus for the nerve ending in Corti's organ. Does the nerve ending record the motion, the velocity or the pressure change set up by the oscillation? Some of the experiments described here seem to indicate that the endings of the acoustic nerve are a pressure recording sense organ. However, other experiments contradict this con-

clusion. It is to be hoped that experiments of this kind will finally furnish convincing evidence for a solution of this important question.

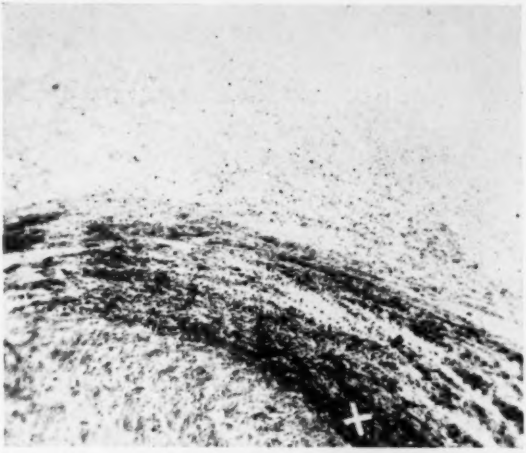

Fig. 7. Degeneration of nerve fibres in primary acoustic centre following limited cochlear lesion (Marchi preparation).

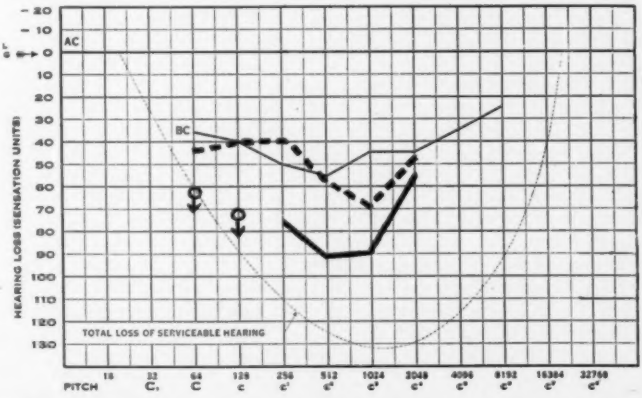

Fig. 8. Function of the cochlea, with destruction of apical coil. The solid line indicates the function by application of sound into the outer air. The broken line is the reflex threshold obtained by application of sound into the middle ear cavity.

Secondly, these experiments seem to show that the end organ is irritable, both by sound entering the cochlea through the oval window and approaching the nerve endings through the scala vestibuli, and by sound waves passing through the round window reaching Corti's organ through the scala tympani. Especially for the cat with its favorable anatomical conditions the direct application of sound into the round window niche could be studied.

Thirdly, it seems evident that the cochlea can be successfully stimulated directly from the air of the middle ear. The action of the ossicles as described in the classical teachings of Helmholtz and Bezold appears in a different light. It is true the inner ear receives the acoustic stimuli normally over the ossicular chain, but the ossicles are not the only and exclusive pathway. A direct transformation of sound waves of the air of the middle ear into the fluid of the inner ear is possible.

University of Chicago.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

III.—ANIMAL INVESTIGATIONS.

(d)—THE ORIGIN OF COCHLEAR POTENTIALS.*†

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The potentials that can be recorded from the cochlea or contiguous structures when sound impinges on the ear have been shown to arise from the cochlea and to be distinct from the action potentials present in the auditory nerve and tract.[1,2]. The particular structures within the cochlea which are concerned in their production and the manner in which they are produced is still undetermined. The small or absent response from cats and guinea pigs with ear infections or with congenital deficiencies in the internal ear has led to the view that the potentials are dependent on the organs of Corti.[3,4] The extensive pathology present in the internal ear of these animals, however, would appear to make it difficult or impossible to ascribe the deficiency to any particular feature of the abnormality. It has, moreover, been found that, in guinea pigs at least, structural integrity of the organs of Corti is not necessarily related to cochlear response.[5] Further evidence for this will be given in the present communication.

The view that some nerve structure is involved in the production of cochlear potentials has come from experimental results obtained following the application of certain materials to the round-window or from their introduction into the cochlea.[6,7] It appears, however, impossible to make such applications and avoid contact of the material with middle ear structures. Even when the material is introduced into the cochlea, overflow of fluid with middle ear contamination is

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difficult to avoid. The possibility that the abolition or great reduction of response found in the experiments reported was due to middle ear rather than cochlear damage has not been excluded. Evidence against the nerve origin of the cochlear potentials, although perhaps not excluding nerve elements within the organs of Corti, is the observation that normal response may be obtained after degeneration of the spiral ganglion and nerve fibres.[4].

The observations forming the basis of the present communication were primarily concerned with the differentiation of middle ear damage from cochlear damage following certain experimental procedures, and the susceptibility of the mechanism developing cochlear potentials to adverse influences. The series includes 72 experiments on guinea pigs under nembutal anesthesia and a few additional experiments on dogs and cats.

Methods: The potential developed by the cochlea as a result of 13 different sound frequencies, from 50 to 4,000 cycles per second, applied to the ear by air or bone conduction was measured in each experiment. The apparatus employed, with the exception of the bone conducting mechanism, has been described.[5] Electrical oscillations from a General Radio audio frequency oscillator were led either to a loud-speaker for air-conducted stimuli, or to an electromagnetic speaker unit acting as a driver to a bakelite rod for bone-conducted stimuli. The experiments were carried out in electrically shielded rooms and the room containing the animal and apparatus used for measuring cochlear potentials was separately shielded from the apparatus generating the stimuli. Air-conducted stimuli were led to the ear through a large rubber tube. For bone conduction, the bakelite rod was placed on the upper teeth of the animal. The output of the oscillator at each frequency was kept constant by the aid of a General Radio output meter. The cochlear potentials, after amplification through a three-stage amplifier, were attenuated if necessary, rectified and measured by a swinging coil galvanometer. Over-all calibration of this apparatus at the different frequencies used made possible the expression of the cochlear potentials in microvolts. In guinea pigs, a needle electrode was placed in contact with the cochlea and the circuit to the amplifier completed by another electrode in the neck muscles. In cats and dogs, the needle electrode was placed near the margin of the round-window. In certain instances, where

none or very small cochlear potentials were obtained, the cochleae were removed for histological study without further procedure. In the remainder, observations were continued as to the effect of experimental procedures on the response to air- and bone-conducted stimuli. Histological examination was carried out in all cases.

THE RELATION OF COCHLEAR RESPONSE TO THE HISTOLOGY OF THE INTERNAL EAR.

In 13 cases of the present series of 72, the presence of middle ear infection was obvious on opening the bulla. Excluding these, there were 43 cases in which the cochlear potentials in response to air- and bone-conducted stimuli were

Fig. 1. Photomicrograph of lower turn of the cochlea of guinea pig 34-59. Right. The organ of Corti and associated structures are normal. The response from this ear was normal.

regarded as normal in magnitude and distribution at the various frequencies. In the guinea pig, maximum response to air conduction occurs at either 200 or 300 cycles, and there is usually a second but lower peak response at 1,400 cycles. Maximum response to bone conduction, under our experimental conditions, occurs at 100 or 140 cycles. In the dog and cat, maximum response in each case is found at much higher frequencies. The maximum normal response from the guinea pig cochlea may be as high as 12 millivolts, but is usually in the range of 3 to 6 millivolts. Responses as high as 20 millivolts have been obtained under certain experimental conditions.

In the 43 cases with normal cochlear response, five were found to have rather extensive pathology in the internal ear involving the organs of Corti and associated structures (see Fig. 2). The organs of Corti were atrophied throughout, Reissner's membrane collapsed and fibrosed, along with the tectorial membrane, to the basilar membrane. In nine other cases this pathological process was found in localized regions, or generally distributed, but of a less advanced stage. No

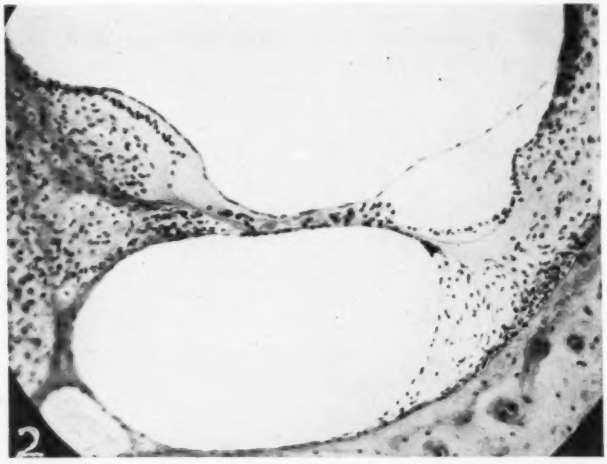

Fig. 2. Photomicrograph of second turn of guinea pig 35-28. Left. Reissner's membrane is collapsed and adhered to the tectorial membrane and organ of Corti. The organ of Corti in this ear is atrophic and fibrosed throughout. In spite of this, the response to both air- and bone-conducted stimuli was normal.

alterations from the normal pattern of response at the different frequencies were found in the cases with localized pathology. In the remaining 29 cases the structures of the middle and internal ears appeared normal.

In 12 of the series of 59 cases in which no middle ear pathology was present, response to air and bone conduction was present, but reduced to varying degrees. In three of these, the pathological changes described above were advanced and involved the whole cochlea; in two, there were regions of localized atrophy, and in the remaining seven there was no evidence of cochlear pathology (see Figs. 3 and 4).

In four cases in this series there was no response, or only traces of response to air- or bone-conducted stimuli at any frequency used. In one of these the organs of Corti and associated structures were atrophic and fibrosed, in another there were localized regions of atrophy, and in the remaining two the cochlea appeared normal (see Table I).

The Effects of Middle Ear Injury: Preliminary experiments of our own led us to question whether many of the effects found following application of materials to the round-

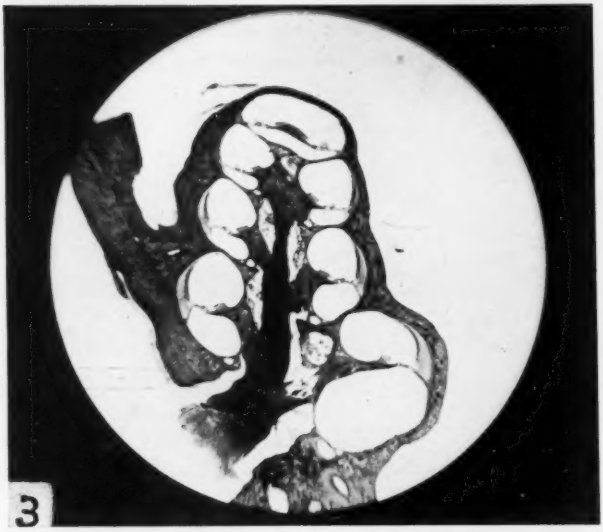

Fig. 3. Low power photomicrograph of cochlea of guinea pig 36-25. Left. Structurally this is a normal cochlea, but the response to frequencies above 300 cycles was very poor.

window, effects which in some cases had been described by others, might not be due to middle ear rather than cochlear damage. It is impossible to apply materials to the round-window with avoidance of possible middle ear effects, and even on direct introduction of material into the cochlea some overflow into the middle ear occurs. For these reasons it appeared important to differentiate between the two. The introduction of bone-conducted stimuli did not entirely solve the problem, for we found that in the guinea pig bone con-

duction response was variable and occasionally as much reduced following middle ear damage as response to air-conducted tones. This agrees, qualitatively at least, with the results of Wever and Bray,[9] who found that following interruption of the ossicular chain in guinea pigs, bone conduction response was reduced, although not as much as the response to air conduction, in about half of their experiments.

Thirteen cases of advanced middle ear infection were found in the present series of guinea pigs. In six, response to air-conducted stimuli was much reduced at all frequencies; in the remaining seven cases it was abolished at all frequencies. In the former group, lower frequencies were more reduced than those in the middle and high range, and five of the six

TABLE I.

RELATION OF CONDITION OF INTERNAL EAR TO RESPONSE TO AIR AND BONE CONDUCTION. THE MIDDLE EAR WAS NORMAL IN ALL CASES.

Number of Observations	Response	Histology of Internal Ear
29	Normal	Normal
5	Normal	Extensive atrophy and fibrosis
9	Normal	Localized atrophy
3	Much reduced	Extensive atrophy and fibrosis
2	Much reduced	Localized atrophy
7	Much reduced	Normal
1	Absent	Extensive atrophy
1	Absent	Localized atrophy
2	Absent	Normal

Total number of observations, 59.

In 23, or 39 per cent, of these the response presents no relation to the integrity of the organ of Corti.

cases showed maximum response at 1,400 cycles instead of the normal maximum at 200 or 300 cycles. In only one of the 13 cases was response to bone-conducted stimuli within the normal range. It was moderately reduced in four, much reduced in five, and absent in three cases. On placing mineral oil in an infected middle ear, response to air conduction is somewhat increased at low frequencies and decreased at the intermediate and high frequencies. Response to bone conduction is increased at all frequencies, especially in the lower range, up to and including 200 cycles, where it may exceed that obtained from the usual normal ear.

Interruption of the ossicular chain in normal middle ears was done in 10 experiments. In six cases response to air-conducted stimuli was absent at all frequencies; in three it was very much reduced, and in one there was moderate reduction. Response to bone-conducted stimuli was abolished at all frequencies in one case; much reduced, although not to the same extent as the response to air-conducted stimuli, in the remainder. Pressure applied to the stapes region of middle ear structures after the chain was interrupted caused marked return of bone conduction response, particularly at low frequencies, while this procedure had little effect on response to air-conducted stimuli. Filling the bulla with mineral oil or mercury caused increased response to bone-conducted stimuli at all frequencies, especially at the lower frequencies (up to and including 200 cycles), where the response may exceed by several times the normal found before middle ear damage was done. The effect of this procedure on response to air-conducted stimuli was much less, but frequently caused return of response at a few frequencies where it had formerly been absent.

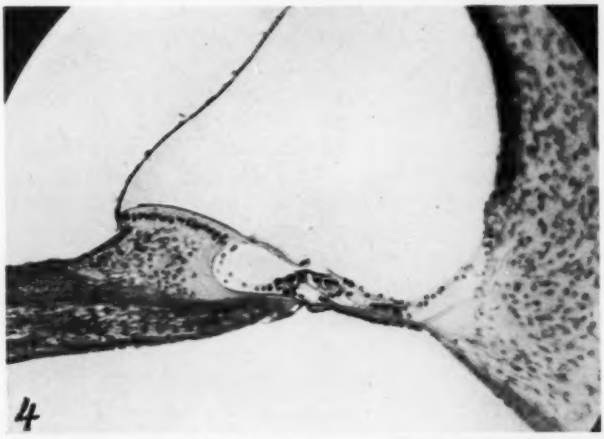

Fig. 4. Photomicrograph of basal turn of cochlea shown in Fig. 3. The organ of Corti is normal.

Injury to the middle ear of the guinea pig, whether from

infection or experimental damage, thus reduces greatly or abolishes response to both air- and bone-conducted stimuli; although the latter is usually less affected than the former. The response to bone-conducted stimuli may be restored, or may even exceed the normal, as a result of altering the physical conditions in the middle ear by applied pressure or by the presence of a liquid. A recognition of these facts appears to offer a satisfactory experimental method for differentiating middle ear damage from cochlear damage.

THE EFFECT OF VARIOUS MATERIALS APPLIED TO THE ROUND-WINDOW OR INTRODUCED INTO THE COCHLEA.

Complete abolition or great reduction of cochlear response to air-conducted stimuli have been reported following introduction of solutions of cocaine,[3,6] quinine[7] and pilocarpine[6] into the cochlea and the application of crystals of cocaine, sodium chloride,[7,8,9] calcium chloride, quinine hydrochloride and glycerine[9] to the round-window. Negative results have been reported for normal saline,[3] distilled water and crystals of glucose.[9] In dogs conditioned to certain tones, application of sodium chloride crystals is reported to cause loss of hearing, as well as abolition of cochlear potentials.[9] It does not appear that in any of these experiments the possibility that the results obtained were due to middle ear rather than cochlear damage has been considered.

It has been shown[11] that many substances placed in the middle ear are absorbed rapidly. Ocular and head nystagmus, turning of the head and body and falling, follow the introduction of certain materials into the dog's middle ear. Cocaine and procaine in 10 per cent solution produced complete labyrinthian paralysis in from five to 25 minutes after application.

On placing materials in the bulla in contact with the round-window, the actual or apparent cochlear response may be altered in several ways, independently of any change in the cochlea itself. In the first place, when dealing with electrolytes, changes in electrical conductivity between the electrodes have to be considered. If the electrode on the cochlea comes in contact with an electrically-conducting solution, the cochlear potentials may be in large part short-circuited. Thus, if the bulla is filled with Ringer's solution until the cochlear

electrode is immersed, response to both air- and bone-conducted stimuli may appear to be greatly reduced or abolished, to return to much larger values when the level of the Ringer's solution is lowered. Another effect belonging under this heading appears to be increased electrical resistance of the walls of the bony cochlea as a result of coagulation, drying or other changes induced by the material in the bulla. In such cases, normal readings are restored when the tip of the electrode is passed through the bone.

In the second place, the material used may alter the physical condition in the middle ear and on the round-window, affecting the transmission of vibrations to the internal ear. The effect of changes in tension of the tensor tympani muscle and of pressure applied to various regions has been studied especially by Crowe and coworkers.[12,13] In our experience, liquids in the normal bulla (Ringer's solution, mineral oil and mercury) increase the response to air-transmitted stimuli slightly at low frequencies and greatly reduce or abolish the response to intermediate and high frequencies. The response to bone-conducted stimuli, on the other hand, is increased throughout; very markedly so at the lower frequencies. Different fluids appear to more or less tune the mechanism to certain frequencies, so far as bone conduction is concerned, and large responses result at these frequencies. Thus, mercury produces a very large response at 70 or 100 cycles, which may exceed several times the normal, while the peak response with less heavy liquids is usually at a higher frequency (140 to 200 cycles).

Finally, the reduction or abolition of cochlear potentials, following the application of materials to the round-window, may result from injury to the structures of the middle ear without the substance having affected the cochlea in any way. It is obvious from our experimental results that the distinction between middle ear and cochlear damage cannot be made from the response to air-conducted stimuli alone and that, in the guinea pig at least, marked reduction or even obliteration of response to bone-conducted stimuli also may result from damage confined to the middle ear. There are, however, certain characteristics which make it possible to distinguish between reduction or obliteration of response due to middle ear damage and that due to cochlear damage. In

the former case, in instances in which response is reduced but not abolished, the response to air conduction is usually reduced to a greater degree than the response to bone conduction. The greatest reduction in air response is at the lower frequencies and in many cases the maximum response is attained at 1,400 cycles, whereas normally the peak occurs at 200 or 300 cycles. Whether the response is reduced or abolished, differentiation between middle ear and cochlear damage as the causative factor may readily be determined by the introduction of some liquid, such as mineral oil or mercury, into the bulla. If the cochlea is undamaged, definite response to bone-conducted stimuli, especially at low frequencies, appears, or if already present the response is greatly increased and may reach or even exceed the usual normal response. This will not occur if the deficient or absent response is the result of cochlear damage.

In the guinea pig, the tip of the cochlea may be opened without greatly altering the response to air-transmitted stimuli, and in a preparation of this sort materials such as crystals may be introduced directly into the cochlea. Overflow of cochlear fluid occurs, however, and in our experience it has been impossible to avoid contamination of the middle ear. The same is true if solutions are injected through a hole in the cochlear wall.

The physical effects produced by the introduction of Ringer's solution, mineral oil and mercury into the bulla have already been described. No permanent damage results and response returns to normal on removal of the material. In 53 instances, various other materials were applied to the round-window membrane, placed in the bulla or introduced through a hole in the tip of the cochlea.

Chloroform was placed in the bulla in 15 ears. The immediate effect observed is the physical change induced by the fluid, a slight increase or no change to air response and a large increase to bone response at low frequencies. At the higher frequencies air response is much decreased, bone response little affected. Within a period of about three minutes the response changes to that typical of middle ear damage. If the chloroform is removed at this time there is is found a marked reduction or near obliteration of response to air

conduction with usually a change of maximum response to 1,400 cycles, and a smaller reduction of response to bone conduction. No further effect occurs if the chloroform is left in the bulla for as long as 40 minutes.

Nicotine (10 per cent) was applied in seven experiments. The results were practically identical with those induced by chloroform and appear entirely as a middle ear damage.

Nupercaine, in 10 per cent solution, in the bulla caused middle ear damage and alterations of response like those produced by chloroform and nicotine. In one experiment crystals of nupercaine were placed inside of the cochlea through the open apex. Fluid from the cochlea overflowed into the middle ear and obviously carried some of the material with it. After 28 minutes, air conduction was found reduced, with peak of 423 microvolts at 1,400 cycles; bone conduction was also reduced. In another experiment an attempt was made to obtain the effect, if any, of nupercaine on the internal ear, avoiding middle ear damage by applying the substance to the internal auditory meatus. After the exposure of the auditory nerve and meatus, about 1/10 gm. of nupercaine crystals was enclosed in a very thin sheet of cotton and packed around the nerve at its entrance. Sufficient serum was present to cause at least a part of the material to pass into solution. Practically no change occurred in response to air- or bone-conducted stimuli during or following the 42 minutes of application.

Alcohol (60 per cent) was placed in the bulla in four experiments. The results were similar to those previously described, indicative of middle ear damage, but somewhat more pronounced. This additional decrease appears to be due to some change in the bony wall of the cochlea increasing the electrical resistance, for in two experiments, in which very low response to air and bone conduction followed chloroform application, insertion of the tip of the electrode inside the cochlea gave responses to air- and bone-conducted stimuli typical of middle ear damage alone.

Formaldehyde (10 per cent) in one instance and picroformalin in two gave results similar to that of alcohol.

Glucose crystals were applied to the round-window in two experiments and caused changes in response typical of moderate middle ear damage, marked decrease in air response, especially at the lower frequency, with change of maximum response to a higher frequency and moderate reduction in bone response. The glucose remained in the bulla for 30 minutes in one case and 38 minutes in the other.

Sodium chloride in crystalline form or in concentrated solution produced, in seven cases, rapid reduction of response to air- and bone-conducted stimuli to zero or near zero in all frequencies. That the cochlea is, however, still capable of large response may be shown by the application of mineral oil or mercury to the bulla after removal of the sodium chloride (see Table II). Crystals of NaCl were applied to the round-window for 41 minutes and then removed. Response

TABLE II.

EXPERIMENT 37-2. EFFECT OF CRYSTALS OF NaCl APPLIED TO THE ROUND-WINDOW.

Frequency	Normal		After 41 Minutes		Same as Last Except Mercury in Bulla	
	A	B	A	B	A	B
50	188	1087	0	107	0	1695
70	1110	1595	0	10	0	1085
100	600	3640	0	254	0	5045
140	2250	554	2	18	0	381
200	2645	148	4	0	0	2
300	3750	10	22	2	2	0
500	2620	16	0	1	1	2
700	2520	26	0	1	1	2
1000	1220	13	0	3	0	1
1404	1035	45	3	3	1	2
2000	34	6	1	3	0	0
3000	94	0	0	0	0	0
4000	0	0	0	0	0	0

The figures under A represent in microvolts the response to air-conducted stimuli, and those under B to bone-conducted stimuli.

to air conduction was reduced to zero or to a trace only over the frequency range. Response to bone conduction was greatly reduced or abolished (peak response at 100 cycles with 25 microvolts, compared to the normal of 3,640 microvolts). On filling the bulla with mineral oil, the response to bone conduction was doubled or more at the lower frequencies, unchanged at the higher frequencies. The mineral oil was

now replaced by mercury. There resulted a large increase in response to bone conduction, exceeding the normal at 50 and 100 cycles. The response to air conduction was not improved. Obviously, the cochlea, although perhaps damaged, was still capable of developing large potentials.

Quinine hydrochloride crystals, applied to the round-window in three experiments, produced results in all cases similar to those described following the application of sodium chloride; a rapid loss or near loss of air response and a great reduction or abolition of bone conduction response. That in these instances the fact that the cochlea is still capable of normal or nearly normal response can be proven by placing mineral oil or mercury in the bulla. Thus, in one experiment (see Table III) showing initially normal response to air and bone conduction, following 40 minutes' application of quinine hydrochloride crystals, response was reduced in each case

TABLE III.

EXPERIMENT 37-1. EFFECT OF QUININE HCl APPLIED TO THE ROUND-WINDOW.

Frequency	Normal		After 40 Minutes' Application of Quinine HCl		Same as Last Except Mercury in Bulla	
	A	B	A	B	A	B
50	607	745	0	0	0	239
70	1480	338	1	1	0	783
100	620	2770	3	3	3	2290
140	3220	1340	9	1	2	160
200	4050	331	19	1	9	14
300	2700	3	84	1	1	15
500	2115	11	1	4	1	1
700	835	9	0	3	1	3
1000	642	2	2	3	0	3
1400	1800	21	1	3	3	6
2000	264	0	1	2	0	0
3000	86	0	2	0	0	0
4000	9	0	0	0	0	0
5000	0	0	0	0	0	0

The figures under A represent in microvolts the response to air-conducted stimuli, and those under B to bone-conducted stimuli.

to zero or to traces of response at the different frequencies. On applying mercury to the bulla, the response to bone conduction was about twice the normal at 70 cycles, and approximately normal at 100 cycles. No improvement in response to air conduction resulted.

EFFECTS OF PASSAGE OF DIRECT CURRENT THROUGH THE COCHLEA.

In four experiments, a polarizing direct current was passed through the cochlea. In the first experiment the current was passed through the electrodes used for measuring cochlear response. A current of 0.8 milliampere at six volts applied for 30 seconds caused no change in response. Two milliamperes at 10 volts produced evident electrolysis and temporary reduction in response. In this ear the cochlea was found normal on histological examination.

In the other three experiments, the current density through the cochlea was increased by an additional electrode on the basal turn of the cochlea, the current being passed between apex and base. Two milliamperes at five volts for two minutes caused moderate reduction of response, associated with evident electrolysis. Recovery following the cessation of current flow was almost complete. No difference was found related to the direction of current flow. Eight milliamperes at 45 volts caused extensive electrolysis, with the cochlea assuming a white milky appearance and an irreversible loss of cochlear response (see Figs. 5 and 6). Extensive damage was found in all turns of these cochleae, fragmentation of the organs of Corti and associated structure, rupture of membranes and coagulation or sedimentation of cochlear fluid.

These results appear to us to offer strong evidence against the nervous origin of cochlear potentials. They also rule out the suggestion[14] that the response depends upon the presence of a polarized membrane in the cochlea.

DISCUSSION.

The mechanism in the cochlea which develops electric potentials as a result of vibrations induced by air- or bone-conducted tone appears extraordinarily resistant to influences which ordinarily exert a deleterious effect on physiological mechanisms. The production of these potentials is also not dependent on the integrity of those parts of the cochlea, the organs of Corti and associated structures, to which evidence from various angles points as the sensory end-organs of the auditory mechanism. There is no valid evidence that the potentials have any relation to nerve action potentials or that they are of nervous origin. The magnitude of cochlear

potentials, their frequency, their correspondence through a wide range with the applied sound frequency, their tendency to spread throughout contiguous tissues and other features serve to distinguish them from the potentials arising in nerves associated with the passage of an impulse.[2] The resistance of the mechanism to toxic substances and other influences that rapidly abolish responses from nerve mechanisms would appear to rule out the possibility that they are related in any way to the nervous structures of the internal ear.

Fig. 5. Low power photomicrograph of cochlea of guinea pig 35-18. Right. Showing effect of passing direct current of 8 milliamperes through cochlea for a period of two minutes.

In a vibrating mechanism such as the internal ear represents, consisting as it does of membranes separating aqueous phases, it is difficult to conceive of electrical potentials from purely physical causes not developing when it is set in vibration. One probable source of potential which is immediately obvious is that produced by the flow of water through membrane pores or other channels of small dimensions, the so-

called flow or streaming potential. We have designed a model which shows that under physical conditions similar to those existing in the internal ear, flow potentials may develop of the order of magnitude and frequencies comparable to cochlear potentials. This model consists of a porous membrane separating two shallow chambers containing water. The outer surface of each chamber is formed by a membrane of oiled silk or similar material. When sound waves impinge on the outer membrane, the vibrations set up cause passage of water

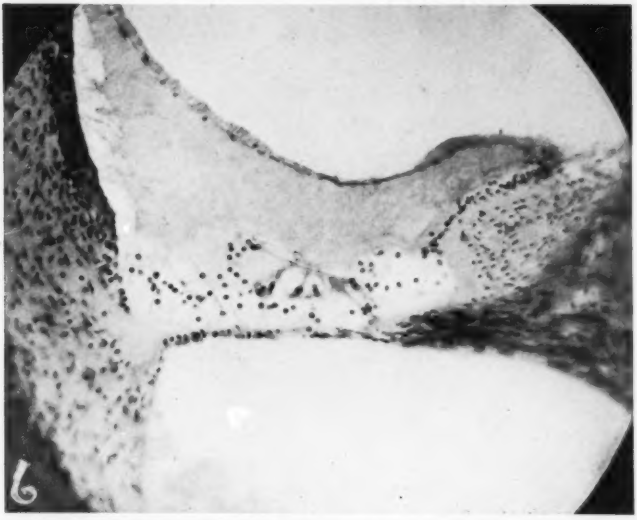

Fig. 6. High power photomicrograph of part of the second turn of the cochlea shown in Fig. 5.

through the porous membrane, and this in turn produces differences of potential between the two chambers at the same frequency as the sound. If the potentials from this microphone are amplified and led to a telephone or loud-speaker, words and speech may be intelligently transmitted, as in the case of the cochlea.

That the cochlear response has the same physical characteristics as that of a microphone has been shown by Covell

and Block.[15] The relation between energy input and output is a linear one until the energy input is increased to the point of overloading.

In a previous work[4] we have shown that the one physical alteration to which the mechanism of cochlear potential response is most susceptible is that of disturbance of the pressure relations between endo- and perilymph. Puncture of the cochlear duct alone produced prompt abolition of response, while opening all ducts into free communication resulted in only moderate reduction of response.

Stevens and Davis,[15] although expressing the view that cochlear potentials arise from deformation of the hair cells of the organs of Corti, find experimentally that the actual potential difference develops across the basilar membrane. When the potential in the scala media, recorded either from a small electrode introduced into the cochlear canal or from an apical electrode, is negative, the potential in the scala tympani, recorded from the round-window, is positive.

CONCLUSIONS.

1. The development of cochlear potentials in response to air- or bone-conducted stimuli does not depend on the structural integrity of the organs of Corti or associated structures.

2. The resistance of the mechanism to adverse influences, including the action of a polarizing current and toxic materials applied to the round-window or introduced into the cochlea, would appear to rule out the possibility of a nervous origin of the potentials or that they result from changes in a polarized membrane.

The response from the cochlea may be simulated by a model in which the potentials arise as a result of flow of water through membrane pores, induced by sound waves. That potentials of this origin must be present when the internal ear is set in vibration appears obvious from the physical structure of the cochlea, and the magnitude and frequency characteristics of such potentials suggest that a mechanism of this type represents the sole source of cochlear potentials.

REFERENCES.

1. Saul, H. J., and Davis, H.: *Trans. Amer. Otol. Soc.*, 1932.

2. Davis, H.; Derbyshire, A. J.; Lurie, M. H., and Saul, L. J.: *Amer. Jour. Physiol.*, 108:311, 1934.

3. Lurie, M. H.; Davis, H., and Derbyshire, A. J.: *Ann. Otol., Rhinol. and Laryngol.*, 43:321, 1934.

4. Van Gilse, P. H. G.: *Acta Otolaryngol.*, 20:23, 1934.

5. Bast, T. H., and Eyster, J. A. E.: *Ann. Otol., Rhinol. and Laryngol.*, 44:57, 1935; and *Trans. Amer. Otol. Soc.*, 1935.

6. Adrian, E. D.; Bronk, D. W., and Philips, Gilbert: *Jour. Physiol.*, 73:28, 1931.

7. Fowler, E. P., and Forbes, T. W.: *Proc. Amer. Physiol. Soc.*, 116:51, 1936.

8. Guttman, J.: The Laryngoscope, 43:983, 1933.

9. Guttman, J., and Barrera, S. E.: *Amer. Jour. Physiol.*, 109:704, 1934.

10. Wever, E. G., and Bray, C. W.: *Ann. Otol., Rhinol. and Laryngol.*, 45:822, 1936.

11. Ross, E. L., and Rawson, R. W.: *Arch. Otolaryngol.*, 22:312, 1935.

12. Crowe, S. J.: Harvey Lectures, p. 100, 1931-32.

13. Hughson, W., and Crowe, S. J.: *Acta Otolaryngol.*, 18:291, 1933.

14. Hallpike, C. S., and Rawdon-Smith, A. F.: *Jour. Physiol.*, 81:395, 1934.

15. Covell, W. P., and Block, L. J.: *Amer. Jour. Physiol.*, 116:524, 1936.

16. Stevens, S. S., and Davis, H.: *Jour. Acous. Soc. Amer.*, 8, 1936.

APPENDIX.

Since this paper was written, two important communications bearing on this problem have appeared. The first of these[1] is concerned with the effect of a sudden change of phase (180°) in a pure tone, applied to the auditory mechanism of cats, on the potentials recorded from the cochlea and from the auditory tract, respectively. The potential waves from the cochlea follow this change in phase immediately, while the tract potentials are nearly or completely suppressed for several subsequent cycles. The suppression is explained by the maintenance, for a period after the phase change, of the opposing forces developed in a resonant structure. Tract potentials are thus regarded as resulting from the vibration of a resonant structure (basilar membrane); cochlear potentials from the vibration of a non-resonant, highly damped

structure. Reissner's membrane is suggested for the latter; a suggestion not in accord with our own findings.

The second communication referred to[2] offers further evidence that the organs of Corti, and more specifically the hair cells and nerve elements associated with this structure, are not essential for the production of cochlear potentials. Following section of the VIIIth nerve in cats, pathological changes of different kinds occur in the internal ear. Normal cochlear response was obtained in several animals in which there were extensive degenerative changes and disintegration of neural elements and of the organs of Corti. This is in agreement with our findings.

REFERENCES.

1. HALLPIKE, C. S.; HARTRIDGE, H., and RAWDON-SMITH, A. F.: *Proc. Roy. Soc.*, Series B. 122:175, 1937.

2. ASHCRAFT, D. W.; HALLPIKE, C. S., and RAWDON-SMITH, A. F.: *Ibid*, 122:186, 1937.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

III.—ANIMAL INVESTIGATIONS.

(e)—AN EXPERIMENTAL STUDY OF THE NEURAL MECHANISM OF HEARING.*†

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The technical difficulties involved in an investigation of this kind invariably make the information obtained rather insignificant in relation to the amount of time and effort put forth. Experiments must be repeated many times before a dependable method can be developed, and even then an inevitably high mortality in the experimental animals persists before consistent success can normally be achieved. The data upon which the following observations are based represents a series of chronic experiments carried out in the Laboratory of Otological Research at the Johns Hopkins in 1934, 14 in number, and a subsequent extensive investigation during the past year in the Otological Research Laboratory of the Abington Memorial Hospital. In the first group, partial and graded division of the auditory nerve was carried out under high binocular magnification. In all experiments partial or complete division of the nerve was considered successful only if the blood supply of the cochlea remained intact.

Hallpike and Smith,[1] and Buttman and Barera,[2] in 1934, reported successful experiments of this type; the former upon one animal with histologic control, the latter in a small series, in which records were made an appropriate length of time following operation. Hallpike and Smith found no cochlea response by the present routine electrical method of examination in their one case of nerve section and spiral ganglion

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atrophy. The second investigators, with similar material, found an active cochlea response following section of the auditory nerve.

In the present series graded division of the auditory nerve was carried out, followed by a relatively equal degeneration of nerve and spiral ganglion cells. By suction a known amount of nerve tissue could be destroyed without damage to the blood supply of the cochlea. The extent of this ganglion cell damage has been determined by actual count in relation to a normal estimated by Howe[3] in his study of the absence of the cochlea effect in albino cats. Regardless of the extent of degeneration of the nerve fibre or spiral ganglion of the nerve and cochlea, the cochlea response has remained practically unaffected. Any impairment shown may be in proportion to the amount of degeneration of the ganglion, but it must be noted (see Figs. 1 and 2) that the degeneration at no point is complete and cannot be interpreted specifically in relation to frequency. In none of the photomicrographs is there any departure from normal in the appearance of the organ of Corti, the cochlear duct or the stria vascularis. Examination does reveal, however, a slightly abnormal appearance of the sulcus cells. This point, though possibly significant, will not be discussed at the moment.

Close examination of these spiral ganglia reveals a crescentic arrangement of the surviving cells which apparently are not entirely related to the specific organ of Corti in immediate anatomic proximity, but are relaying nerve elements to the more remote or apical spiral ganglia. Fig. 3 shows a normal apical ganglion with marked reduction in the number of ganglion cells towards the base. By actual count the total number of ganglion cells in this cochlea was reduced below normal by half and yet the cochlea response was entirely unimpaired.

Protocol 1: Experiment No. 876 (Johns Hopkins),*. Nov. 30, 1934: Auditory nerve partially divided by suction, left. *Anesthesia:* Intratracheal ether. Started 10:00 A.M.; ended 11:45 A.M.

*Penfield has described a method whereby brain tissue, whether normal or pathological, may be removed by suction with a minimum of trauma to adjacent structures. This procedure has proved of great value whenever ablation could be carried out under direct observation and is the only method by which controlled damage of intracranial structures can be produced.

Procedure: Operative field cleaned with iodine and alcohol. A curved skin incision was made over the occipital region, the temporal muscle freed from its attachment and a small trephine opening made in the skull over the lateral cerebellar lobe. This opening was extended downward and the dura then opened. A small portion of the lateral lobe of the cerebellum was removed by suction. The auditory nerve was almost completely divided, leaving only a thin strand intact, and the blood supply was preserved. Wound was closed with

Fig. 1. Midmodiolar region of cochlea of cat after auditory nerve had been partially divided and generation of ganglion and nerve had taken place. Note arrangement of surviving ganglion cells and almost complete absence of nerve fibre in the osseous lamina. The organ of Corti is entirely normal.

fine, black silk. The animal was in excellent condition at end of operation.

Dec. 1: Animal in very good condition. No ataxia or nystagmus.

March 5, 1935: *Anesthesia:* Intratracheal ether. Started 9:35 A.M.; animal sacrificed 11:45 A.M.

Procedure: Animal prepared for test in usual manner, silver wire electrodes being placed on both auditory nerves.

Ground electrode placed in neck muscles. Both bullae were opened. The incision over the left cerebellum had healed very well and it was quite simple to insert the electrode through the opening previously made in the skull. Results:

The cochlear response in this animal was entirely normal for all frequencies tested from 128 to 8,000 d.v. The measurements were made by the acoustic balance method, which has been described previously.

Autopsy: Intracardiac injection done; left ear and brain stem saved for sectioning. Both ears were carefully removed and the cerebellum and brain stem cut a little to the right of the midline. The lateral lobe of the cerebellum on the left side was perfectly normal. In the mouth of the internal canal the nerve appeared to be definitely scarred, although

AIR CONDUCTION LEFT EAR (OPERATED SIDE).

Electrodes	128	180	250	500	1000	2000	4000	8000
Nerve	22	19	24	20	20	30	39	54
Pickup test	....	....	....	....	....	....	....	*
Cochlea**	30	20	20	23	20	20	37	40
RIGHT EAR								
Nerve	....	27	30	27	40	41	67	....
Cochlea	....	30	33	35	33	35	43	....

*Too faint to balance.

**Silver foil was placed in the round-window and used as a cochlea electrode. Because of the high intensity of the cochlea response it was necessary to reduce the input control by five.

it had not been completely divided as far as one could tell from gross examination. The nerve was left intact and the brain stem on this side was also preserved, including the colliculi. The electrode had apparently been inserted into the scar tissue. The artery could not be seen. Both ears were perfectly normal, with no sign of infection.

In the later group of experiments a technique has been developed whereby isolated ablation of specific units of the central auditory pathway can be carried out. Among the ablations proven by gross and microscopic examination, the cochlea ganglia separately and together have been destroyed and isolated lesions of the tuberculum acousticum produced. These lesions have, of course, been unilateral in every instance. Following the production of the lesion, sufficient time

was allowed to elapse before final testing and preparation of the material for microscopic study of degeneration. As stated above, a large series of such experiments have been carried out. Technical difficulties and high operative mortality accounts for the fact that half of this group of approximately 80 experiments must be regarded as failures. The procedure employed in all successful experiments is as follows:

Anesthesia: Urethane, supplemented by intratracheal ether.

Operation: The usual ventral midline incision of the neck was made with section and suture of the transverse jugular

Fig. 2. Adjacent ganglia in same animal. Note that ganglion cells appear normal in opposite positions. Slight atrophy of sulcus cells.

vein. The sternomastoid muscle, carotid artery and nerve are retracted laterally, exposing the medial surface of the bulla, and the trachea and esophagus are retracted medially to expose the entire area in the midline between the two bullae. The two longus capitis muscles are then freed from their origin on the basal plate of the skull and retracted caudally. A small trephine is then made in the basilar plate with a fine burr and this opening enlarged to give wide exposure of the lateral cerebellar artery and vein, the origin of the VIth nerve, the lateral pyramid, and the trapezoid body.

The opening may be carried across the midline if desired, with consequent exposure of the basilar artery. The dura is then opened and cerebrospinal fluid removed by suction until it no longer obscures the field of operation. The trapezoid body and the olive beneath its surface are thus immediately exposed for ablation. This ablation is carried out with a glass-shielded cautery point, the shielding leaving exposed only the cross-section of the cautery needle. With a specially curved needle, the dorsal, or ventral cochlear nucleus, or the tuberculum acousticum, can be scorched, either separately or

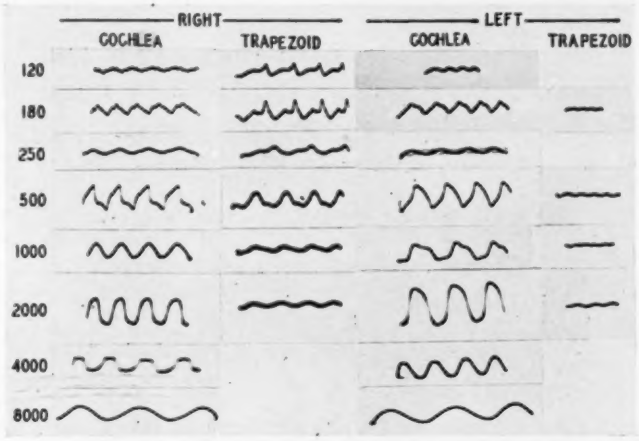

Fig. 3. Illustrating normal nerve and ganglion at apex only. The cochlea effect in this animal was entirely normal for all frequencies 120 to 8,000 d.v. The ganglion cell count was less than half the normal number.

together. If bleeding occurs, this is readily controlled by gentle packing. The opening in the basilar plate is then covered over with a piece of muscle and the wound closed, layer by layer, with silk sutures, and the skin with continuous fine, black silk.

After recovery from the anesthetic, these animals show all degrees of a combined cerebellar and vestibular disturbance, depending upon the severity of the lesion. With proper care, however, they make a good recovery and are in excellent condition after the period of degeneration has elapsed.

For final testing the animal is again anesthetized, the operative wound opened and both bullae exposed to give access to the round-windows. In this second procedure care must be taken to prevent the escape of cerebrospinal fluid. Rapid loss of the fluid in this particular location causes a marked falling off in the response, both from the cochlea and the auditory tract, due to the proximity of the cochlear aqueduct and loss of intracochlear fluid through it. Reported observations of the rapid decline of response from electrodes placed in the brain stem are probably due to this factor. An insulated

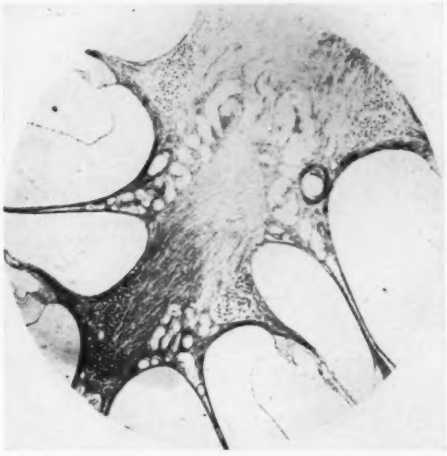

Fig. 4. Note similarity of cochlea response on two sides. On the left, operated side, there is no action current wave in trapezoid body response, while it is marked on the right, normal side.

platinum electrode is inserted through the dura on the unoperated side and a measured distance through the muscle on the operated side into the two trapezoid bodies. Cochlea electrodes are also placed in identical positions on the two sides.

Both acoustic and oscillographic records can then be made with the electrodes in these four different positions. After records have been obtained the animal is sacrificed and an intracardiac injection of normal salt solution, followed by

10 per cent formalin, is made. At autopsy the position of the lesion is determined grossly and the placement of the trapezoid electrodes checked. The brain is then prepared for Marchi staining and sections made. A protocol of such an experiment follows:

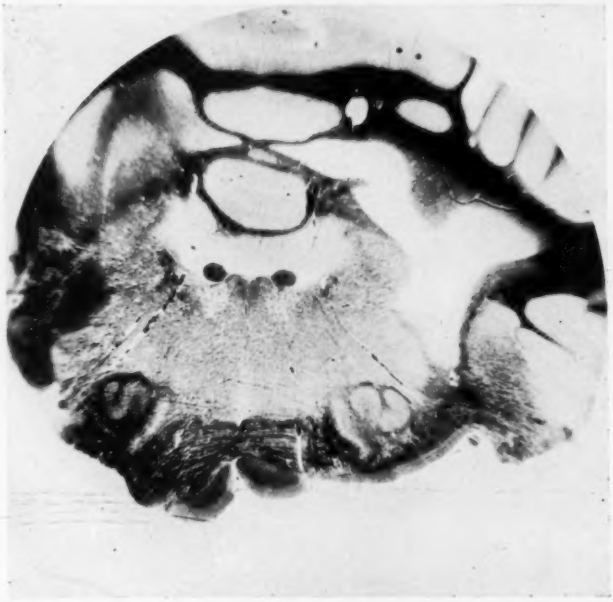

Fig. 5. Marchi stain showing degeneration following cautery ablation of the dorsal and ventral cochlear nuclei on the left side. Note particularly that the left olive and paraolive are completely uninvolved. The cochlea response was entirely normal, while currents in the left trapezoid were absent.

Protocol 2: Experiment No. 228 (Abington), April 14, 1937: Ablation cochlear nuclei, left. *Anesthesia:* Intratracheal ether. Started 10:30 A.M.; ended 12:15 P.M.

Procedure: Operative field cleaned with alcohol and iodine, and the usual midline incision was made. The left bulla was exposed and the base of the skull just medial to the edge of the bulla freed of its muscle attachments. A hole was drilled through the skull just medial to the edge of the bulla

and enlarged with a sphenoid punch. Very little hemorrhage occurred, and this was quite easily controlled. The dura was opened and the basilar artery could be seen. A glass-insulated cautery electrode was introduced in the proper position and scorching of the cochlear nuclei carried out. Wound closed with fine, black silk after placing a piece of muscle over the opening in the skull. Animal in good condition at end of operation.

Fig. 6. (After Victor Mann.) In this drawing the point of ablation appears on the right, "vent. g." Details of the degeneration, not apparent in the photomicrograph, are clearly shown.

Recovery Note (3:30 P.M.): Deep reflexes active and equal. Respiration, 60; pulse, 130. There is mild vestibular-cerebellar syndrome with nystagmus to the left. Animal has fine tremor and deviation of the head, with falling to the left.

April 16: Animal recovering rapidly. The only residual symptoms are slight deviations of the head and a tendency for the pelvic girdle to fall in either direction, principally to the left.

April 19: Animal has entirely recovered.

April 23: *Anesthesia:* Intratracheal ether. Started 9:45 A.M.; animal sacrificed 11:40 A.M.

Procedure: The trachea and esophagus were carefully dissected free from surrounding structures, cut across and the end of each tied. This gave a free exposure of the base of the skull after the right longus capitis muscle was freed from its origin. The left had been previously freed at time of operation. The right side was then carefully drilled in the usual manner. The dura was not opened until the electrode was inserted in an attempt to overcome loss of cerebrospinal fluid which has been rather profuse in the preceding experiments. Both bullae were opened and the cotton-covered cochlea electrode placed in the right trapezoid body after a pin hole had been made in the dura. Ground electrode placed in neck muscles. After photographs had been made of the oscillographic recordings from these electrodes they were placed in a similar manner on the left, the operated side. Intracardiac injection done; brain and ears saved for sectioning.

Autopsy: 1. A left-sided lesion from ablation, 0.5 mm. in size, irregular, cephalolaterad to and involving the ventral nucleus of the cochlear nerve. *2.* Left side mid-trapezoid, 0.5 mm. circular lesion due to placement of electrode. *3.* Right side just lateral to pyramid, but within the trapezoid body about midway cephalocaudad, 0.5 mm. circular lesion due to electrode placement.

Acoustic measurements of the cochlea response were made. For the purpose of graphic representation an oscillograph record (see Fig. 4) illustrates the cochlea and trapezoid body responses in this experiment. The former are typical and normal on the two sides. On the operated side, leading from the trapezoid body, there is a complete absence of action current in the wave, responses being obtained only for the frequencies 180, 500, 1,000 and 2,000 d.v. On the normal side action currents for the frequencies 120, 180, 250, 500 and 1,000 are apparent.

Mann, working in our laboratory, has made an exhaustive study of the degeneration of the auditory tract following these specific ablations. The actual anatomy of the auditory pathways as determined by Marchi stain will be reported later. Photomicrographs and drawings (see Figs. 5 and 6) (Mann)

of this particular experiment illustrate the degeneration following ablation of the two cochlear ganglia. The remarkable contralateral representation is immediately apparent and is, of course, well known. The less obvious manifestation of this relationship, as indicated by degenerative changes in the decussations of Monokoff and Held, is of possible interest. More striking and of greater significance is the complete isolation of the olive and paraolive, so far as their connection with the afferent auditory tract of the cat is concerned. This fact has been demonstrated repeatedly and unequivocally.

DISCUSSION.

Examination of the electrical response in the ears of cats upon which graded division of the auditory nerve has been carried out indicate that the cochlea response is independent and unrelated to either the presence or absence *in toto* or in part of the neural elements of the cochlea. Action currents, however, by their presence or absence following ablation of specific regions of the central auditory pathways, controlled by cochlear ganglion and auditory tract degeneration, must be the standard by which true auditory response in relation to the sense of hearing is to be measured.

Separation of the cochlea and action current response would seem to be of greater significance than examination of either one to the exclusion of the other. Theoretically, an experimental bilateral elimination of action currents should result in the production of a clinically deaf animal. In such an animal the cochlea response should remain unimpaired. It is hoped that this contention will be demonstrated promptly. Up to the present time absence of the organ of Corti, whether congenital or atrophic, is the only condition in which the cochlea effect is consistently absent.

CONCLUSION.

1. Degeneration of the spiral ganglion of the cochlea, following graded section of the auditory nerve, does not affect the cochlea response as measured by electrical potential estimation.

2. Ablation of the dorsal and ventral cochlear nuclei, with subsequent demonstration of peripheral and central neural degeneration, has no effect upon the cochlea response in the ears of cats.

3. Under identical conditions action currents are eliminated following ablation, even though the cochlea response is entirely normal.

4. In the cat, the intact organ of Corti, regardless of nerve or ganglion, can initiate its individual response.

5. Though action currents may well be a part of the total cochlea response, the opposite is not the case.

6. The chronic experiment—sterile operation and subsequent examination—is the only sound method of studying the neural mechanism of hearing.

BIBLIOGRAPHY.

1. HALLPIKE, C. S., and RAWDON-SMITH, A. F.: The "Wever and Bray Phenomenon." A Study of the Electrical Response in the Cochlea, with Especial Reference to Its Origin. *Jour. Phys.*, 81:3:395, 1934.

2. GUTTMAN, G., and BARRERA, S. E.: Persistence of Cochlear Electrical Disturbance on Auditory Stimulation in the Presence of Cochlear Ganglion Degeneration. *Amer. Jour. Phys.*, 109:4, 1934.

3. HOWE, H. A.: The Relation of the Organ of Corti to Audioelectric Phenomena in Deaf Albino Cats. *Amer. Jour. Phys.*, 111:187, 1935.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

IV.—EXPERIMENTAL CLINICAL AND DIAGNOSTIC METHODS.

THE DIAGNOSIS OF NERVE DEAFNESS.*

DR. S. J. CROWE, Baltimore.

This will be a valuable meeting if it does nothing more than direct your attention to the extent and complexity of the auditory apparatus and leads you to think of the ear as something more than the tympanic membrane, Eustachian tube, ossicles, mastoid and cochlea. The otologist is the expert who is depended on to recognize and treat the disorders of the ear; we are reasonably successful with the acute and chronic suppurations but know very little about the cause, the recognition or the treatment of impaired hearing. Hearing acuity plays an important part in economic and social life, and the fact that so little is known about deafness should fire every otologist with the ambition and determination to aid in the advance of this branch of his profession. Before the otologist can do his part, however, he must have special instruction in the embryology, anatomy, physiology and pathology of the ear and its central pathways. A national move in this direction is indicated by the higher standards imposed each year by the American Board of Otolaryngology. We, as otologists, must be able to visualize and think of the hearing apparatus as a whole: the structures in the temporal bone, the auditory pathways in the brain stem and cortex, the relation of these pathways to other nerves and, finally, the association fibres which connect the temporal lobe with other parts of the brain. As our scientific knowledge advances, new methods for clinical examination will be devised. Advance will come more rapidly if the young otologist is prop-

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erly trained; if he is encouraged to show an interest in deaf patients and the cause of deafness; if he will keep himself informed about the newer instruments of precision and employ standardized methods for testing the hearing, so that his results may be compared and pooled with the experiences of others. A move toward the standardization of tests was made by this Society, in 1936, when the report on Methods of Testing the Hearing by Bone Conduction was presented at the Detroit meeting. The electrical method of study, introduced by Wever and Bray, and the experiments on conditioned animals are most important and stimulating. The

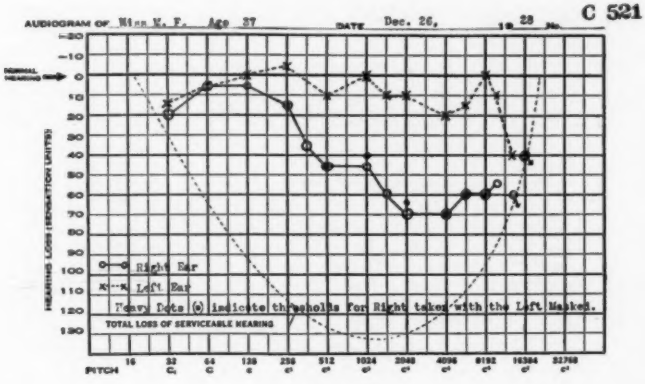

Fig. 1.

ultimate object of all experimental work on the ear, however, is to suggest new ideas to the clinician, who, after all, is the most important link in the chain.

I will limit my remarks to one phase of the subject under discussion—*Bone Conduction.* The object of the clinical test for bone conduction is to determine the sensitivity of each ear separately. When a vibrating tuning fork or bone conduction receiver is held on the mastoid process, the entire skull vibrates with the same frequency. Both cochleae are thus stimulated and the results of the test are often wholly inaccurate and confusing unless the opposite ear is masked.

The better hearing ear must be masked when testing for bone conduction acuity.

This is equally true for air conduction, even when the tests are made with the audiometer at threshold acuity. Two audiograms (see Figs. 1 and 2) illustrate this point. The cause for the impaired hearing on the right side was a small acoustic tumor, which was just beginning to compress the nerves in the internal auditory canal (see Fig. 1). In this patient a second examination with the left ear masked showed that the original test was correct. The audiogram shown in Fig. 2 indicates moderately impaired hearing on the left side, but

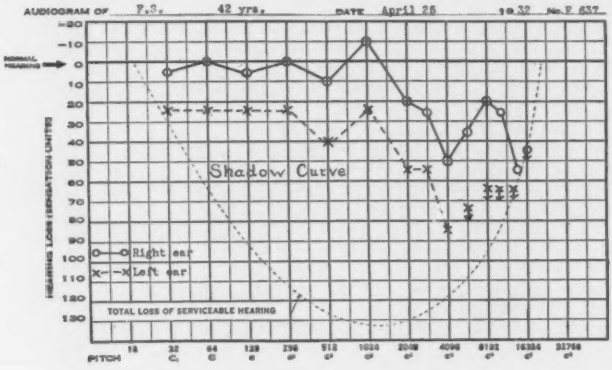

Fig. 2.

when the patient was tested with the opposite ear masked he was found to be totally deaf in the left ear. Operation disclosed a large acoustic neuroma on the left side. In all patients with normal hearing in one ear and partial or total deafness in the other ear, it is absolutely necessary to use masking in order to make an accurate test for either air or bone conduction acuity.

A simple, inexpensive instrument, such as that described by your committee in the *Transactions of the American Otological Society*, page 74, 1936, is very satisfactory for masking purposes. A rheostat regulates the intensity, and calibration enables the observer to make subsequent tests

under the same conditions, so far as the masking sound is concerned. It is true the masking noise may cut down the number of seconds a tone is heard by air or by bone, but this in no way interferes with a comparison of the hearing acuity by air and by bone on the two sides.

During the past 10 years, Dr. Walter Dandy has divided the entire auditory nerve or only the vestibular division of this nerve for the relief of vertigo (Méniére's symptom complex) in 175 patients. In addition, we have examined a large

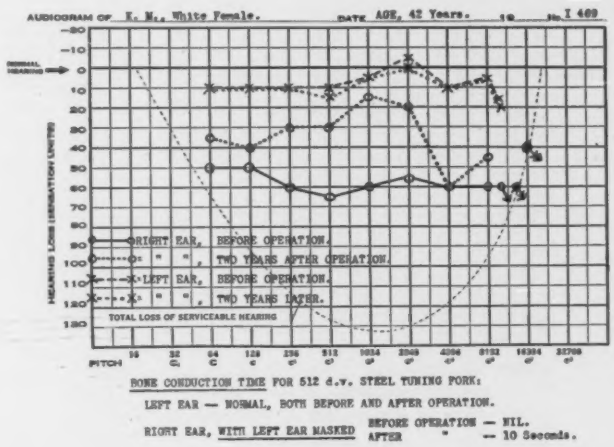
AUDIOGRAM OF E. M., White Female. DATE AGE, 42 Years. No. I 469
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
RIGHT EAR, BEFORE OPERATION.
" " , TWO YEARS AFTER OPERATION.
LEFT EAR, BEFORE OPERATION.
" " , TWO YEARS LATER.
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
BONE CONDUCTION TIME FOR 512 d.v. STEEL TUNING FORK:
LEFT EAR — NORMAL, BOTH BEFORE AND AFTER OPERATION.
RIGHT EAR, WITH LEFT EAR MASKED BEFORE OPERATION — NIL.
AFTER " — 10 Seconds.

Fig. 3.

number of patients with so-called pseudo-Méniére's disease. Otoscopic, tuning fork and audiometric tests were made on all of these patients before and after operation. We have followed up and re-examined 75 of the patients whose auditory or vestibular nerve had been divided from one to nine years previously. The time schedule does not permit an analysis of the results, but I will refer briefly to some findings that have a direct bearing on the subject of this symposium.

The usual symptoms of Méniére's disease are: vertigo, tinnitus and impaired hearing. The impaired hearing is of the nerve type; *i.e.*, air conduction greater than bone conduction;

the Weber test is referred to the better hearing ear and bone conduction is either much shortened or entirely absent. In other words, these patients have all of the classical signs of an organic nerve lesion, but repeated examinations have shown that the hearing acuity may fluctuate. The extent of the improvement by air conduction is illustrated in Figs. 3 and 4. We have also observed a variation in bone conduction acuity from total loss at one examination to normal at a subsequent test one or two years later. In making these tests every precaution was taken to insure accuracy. We do not

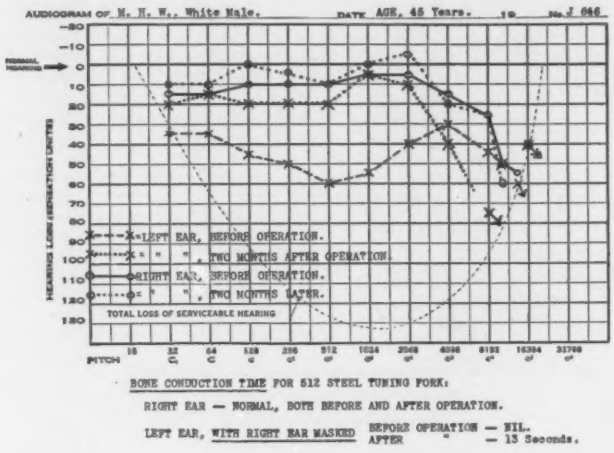

Fig. 4.

know either the location or the pathology of the lesion which gives rise to these symptoms, but we must conclude that even a total absence of bone conduction on one or both sides does not necessarily indicate irreparable damage to the inner ear or nerve.

The second point is the tinnitus. We will discuss tinnitus at this time only in its relation to the accuracy of the tests for bone conduction. In patients with Ménière's symptom complex we do not know the location or the pathology of the lesion giving rise to the tinnitus, but we do know that the lesion is often located in the central auditory pathways.

For example, in 20 patients in whom tinnitus was a prominent symptom, only eight were entirely relieved of this symptom after a total division of both the cochlear and the vestibular nerve; the tinnitus was unchanged in 11 and was much worse after the operation in one patient. The average time between the first and the last examination in this group was four and one-half years. It is inconceivable that any cochlear lesion should continue to give rise to tinnitus after total division of the nerve.

It is thought by many that loud tinnitus acts as a masking noise and the impairment or loss of bone conduction acuity is due to this factor. Among 28 patients re-examined on an average of two and one-half years after the division of the vestibular branch of the auditory nerve alone, the hearing by bone conduction became progressively worse or remained unchanged in nine, although in six of these nine patients the tinnitus entirely disappeared soon after the operation. Four patients with a total loss of hearing by bone conduction never had tinnitus at any time, either before or after the operation. Moreover, the study of 100 patients shows that loud, distressing tinnitus in one ear does not impair the bone conduction acuity in the opposite ear; therefore, unilateral tinnitus does not mask the hearing by bone conduction in the opposite ear. These observations show that tinnitus, when present, is a symptom of the lesion causing impaired hearing by bone and by air, but is not the cause.

Our functional tests and the findings at operation on patients with Ménière's symptom complex have convinced us that the symptoms are not due to pressure changes in the cerebrospinal or intracochlear fluids, and that the lesion is probably in the nerve or the auditory pathways in the brain. It is of the greatest importance for the advancement of our knowledge of this disease and of many allied conditions that postmortem studies be made on patients who have been accurately tested, and that such studies include the central auditory and vestibular pathways, in addition to the structures in the temporal bone. We think the type of lesion responsible for the symptoms of Ménière's symptom complex may give rise to a great variety of other symptoms, depending on its location and extent. Some patients may have attacks of vertigo, deafness and tinnitus; others vertigo alone. We

all see patients with an unexplained loss of vestibular function, severe tinnitus, or impaired hearing of the inner ear type without vertigo or other symptoms. Some patients with so-called Méniére's disease have a loss of consciousness lasting from a few minutes to an hour, transient diplopia, and sometimes impaired sensitivity of the cornea on the affected side. When we understand the nature of the lesion responsible for the symptoms of Méniére's symptom complex we will then be able to understand and diagnose correctly many of the less severe but common disorders of the auditory and vestibular apparatus.

Johns Hopkins University.

SOME PSYCHOLOGIC ASPECTS OF DEAFNESS.*

DR. PHILIP PIKER, Cincinnati.

I would like to emphasize that the psychologic reaction of an individual to a given situation depends much more on the individual than it does on the specific situation. John Brown will respond differently to a certain set of circumstances than will James Smith. The reasons for the difference in responses result from the differences in the personalities of Brown and Smith; therefore, it is somewhat misleading to speak of the psychological aspects of deafness, as though deafness in itself were possessed of a psychology and could be depended upon to provide the same sort of reaction in everyone whom it touched. Some of us behave thus and so as the result of the loss of a limb, or the sudden accumulation of wealth; while others exhibit entirely different patterns of behavior under similar conditions. The personality of each human being is an exceedingly complex thing, and no two personalities are exactly the same. It is this personality of ours with which we attempt to adjust to life, to find contentment, to achieve a measure of success. Because our personalities are so varied, our efforts to adjust are laid along different lines. What is desirable to one individual is unimportant to the next. Each of us measures happiness in his own terms; and likewise, disappointment, frustration, and unhappiness are interpreted and evaluated by people in manners characteristic of themselves.

Though our personalities vary with each of us, there is one aspect of our existences that may be considered generally standardized. Most of us are endowed with physical set-ups which are more or less alike. Despite the fact that some of us are tall and some short, some fair and some dark, some fat and some lean, the similarity in our physiques is sufficiently marked to cause those of us who occasionally think about it to marvel at the consistency of our physical standardization. As part of this similarity of physical attri-

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bute, we have in common our powers of perception—the senses of hearing, sight, smell, taste, and touch. These senses make up the equipment whereby we familiarize ourselves with the world about us; they enable us to be aware of what is going on, to gather impressions from which we may formulate opinions, to make apparent what might be desirable, and to warn us of what might be distasteful or dangerous. The senses, then, are tools that we use in helping us to adjust; but it is important to realize that they are merely tools—a means toward an end. It is the goal that is most important, and not the tools with which we attain that goal. The goal is adjustment—happiness—a life that is adequate, according to each individual's own standards of adequacy. The question that arises, always, is whether or not it is possible to make a satisfactory adjustment with defective tools. I shall not waste your time answering that question in detail—you know that it is not only possible, but more and more frequently an accomplished fact. But isn't it extremely difficult? It is only as difficult as each individual allows it to become. Permit me to remind you again at this point of the differences that exist in personalities. All of you know people who are unduly unhappy, neurotic, or easily upset over circumstances which you feel that you could take in your stride. They have been unable to adjust to these circumstances as well as you might because their personalities (which mean their emotional make-up, their senses of values, their attitudes toward life, their senses of humor, etc.) are not as stable and adaptable in this particular set of conditions as yours might be. The question, then, is not whether or not an individual can adjust to a difficulty, but whether or not he can adjust to life in spite of that difficulty. The answer to that question cannot be given as a general rule—the answer depends on the individual who is seeking it. The adjustment can be made; whether or not an individual will make it depends on that particular individual's attention, his sense of values—his personality generally.

What, now, are some of the undesirable psychologic reactions which may occur in an individual with impaired hearing? I am not saying that these reactions must inevitably occur, or that they always occur. These are the more commonly seen reactions in only those individuals who are permitting their deafness to discolor some part of their adjust-

ment to life. In considering the effects of deafness on an individual's personality, it would be well to divide the problem into two parts, one dealing with those individuals who have been deaf since birth or from very early childhood, and the other having to do with deafness which developed later in life (after the individual has had the use of his faculty of hearing for a time.) It will hardly be possible, however, to discuss the problem from these two angles in this short paper. Let me merely attempt to treat the subject generally, and to point out a few of the more frequently noted psychologic reactions which may accompany deafness in general.

Consider first that trait which all of us have in greater or less degree, pride. It is a part of each of us, and all of us cater to it to some extent. With some of us it assumes exaggerated importance, and we become overly independent and stand-offish in order to inflate it. With others, an attempt is made to feed it by gaining everyone's tolerance and good will, and excessive humility is the result. Still others, in an attempt to nurture their pride, are constantly calling attention to themselves, their achievements, their abilities; and the result is usually termed a show-off. And so it goes with those of us who are too servile to our pride. Even the rest of us, though, have a sensitivity to the demands of our pride, and are frequently hurt and defensive when it is threatened. With many individuals, a sensitive point in regard to pride is physical make-up and efficiency. Some short men are sensitive about their size. Many stout women go to all sorts of extremes to reduce the size of their figures. A sore point with some is lack of strength, or a deformed part of the body, or freckles, etc. The same thing often holds good for deafness. To some individuals, to be deaf is to lack something that others have; it detracts from perfection, and sets one off as being different, incomplete. An individual of this type often will not admit, even to himself, that his hearing is beginning to be less acute. He will readily grasp and believe any explanation he can create to excuse his not hearing as well as he should: wax in the ears, too much noise around, others are not speaking loudly enough, a cold, etc.

What are the likely results of this sort of reasoning on his part? The first, and most serious, is that having con-

vinced himself that his hearing is fundamentally good, he will not consider it necessary to avail himself of medical attention that might help him, or of aids to hearing that might increase his efficiency and comfort considerably. Secondly, he will be under more and more tension as the result of unconsciously straining to hear better—a constant strain which, like all constant strains, will tend to make him emotionally tense, easily fatigued generally, and irritable. Third, because he doesn't hear so well, he will misunderstand statements that are made, follow instructions incorrectly, and make what seem to be pointless and often silly replies in conversation. And fourth, with things not going so well, and because all of us have pride and don't like to admit our own deficiencies, he may soon fall into the habit of blaming others for his own mistakes. This last development is known in psychiatry as projection—the projection on to someone or something else of the blame that should be attached to one's self. All of us project to some extent occasionally. For example: I walk into my room at night, and the light is not on. In the darkness I stumble over a footstool that is standing precisely where I put it when I left the house earlier in the day. I hurt my shin, lose my temper—and what happens? I kick the footstool clear across the room. This is an example of projection. I am hurt—I need to vent my emotions on something, and am not in the habit of taking such things out on myself. Without thinking, I blame the footstool for the situation and proceed to punish it, when, actually, if the blame is to be placed anywhere, I should accept it myself for putting the footstool where it was, or for not turning on the light. As I said, all of us do this sort of thing to some degree. The tired business man, after a bad day at the office, comes home and finds fault with his wife's cooking. And so on. These are occasional occurrences in the life of every individual; and because they do not make up the greater part of the individual's activities or attitude, are tolerated and usually do not make for significant unpleasantness. But with the man who is hard-of-hearing, and at the same time overly sensitive and proud, there exists a condition which is continuous; and the danger of projecting excessively is a real one. He may be irritated because he thinks people lower their voices when talking to him, thus deliberately making it hard for him to hear. He may feel that people look sur-

prised or laugh at his replies because they don't like him, or are trying to make him appear ridiculous. His decreasing efficiency at his work and his mistakes may be attributed to a desire on the part of his fellow-workers or employers to get rid of him. He may even find in his home what he thinks is evidence that his family is turning against him, considers him a load, etc.

This is one of the psychologic reactions which can, and frequently does occur in individuals who have defective hearing. It may occur in those who refuse to admit even to themselves that their hearing is impaired; and it may also develop in individuals who recognize their hearing difficulties but who have permitted these difficulties to discolor their thinking and adjustment generally.

Another reaction which we see is the development of timidity, shyness, self-consciousness. Particularly is this likely to occur in individuals whose personalities tend to be of the withdrawing type, even without the factor of deafness. Some people (because of their early training, or illness, or a number of other possible influences) have more than the average feeling of insecurity. They don't quite feel that they can cope satisfactorily with the problems of living. Feelings of inferiority are apt to flourish in individuals of this type. Superimpose on this existence of excessive inferiority feelings (I say excessive, because all of us have some)—superimpose the factor of deafness, and these individuals are apt to have their feelings of inferiority and insecurity accentuated to a marked degree. As a result they may react in one of two ways: Some will become even more shy and timid, will have less faith in themselves, will tend to withdraw from society more and more in order to save themselves hurts, and hate what in their own thinking have grown to be great deficiencies; or they may rationalize their situations to the extent that they are not consciously sensitive to their feelings of inferiority, and have convinced themselves sincerely that life is more attractive away from society generally. Others may react to their feelings of inferiority in just the opposite direction. They may, in trying to hide their supposed inferiorities, overcompensate by becoming overly active, bullying, officious, impertinent, etc. They may push their way into conversations and gatherings uninvited, or seek to

monopolize attention. Sometimes, in an effort to show both themselves and others that they are in no way handicapped, they will attempt to do work which they are unfitted for definitely, and thus foolishly attract more attention than is necessary to themselves and their limitations. This is the phenomenon of overcompensation, which you may see all about you if you are a keen observer and which is by no means confined only to some of the deaf. It is more apt to occur in the so-called extrovert than in the introvert (or withdrawing) type; but it may be seen developing in both.

Other reactions include the development of self-pity, a particularly crippling emotion, and one which is likely to do the individual far more harm than the deafness itself. Of the same material is woven the feeling of bitterness that some people allow to grow within themselves—a bitterness that is directed at those with better hearing than themselves, and against life in general. There is hardly any emotion which is as likely to discolor an existence as is bitterness. Its source in most instances is not difficult to find, psychologically. It represents the reaction of an individual who is in the first place unable to face an issue, and in the second place unable to admit to himself that he can't face it. The result is a twisted attitude in which he obtains some comfort from placing the blame for his misfortune on an unjust world.

Let me point out at this time the existence of a psychologic factor which, in my opinion, is universal and against which all of us, whether or not we realize it, must fight in our effort to adjust on an adult level. I refer to the tendency in all of us toward being dependent. We are born into an environment which cares for infants and children. In the beginning we are fed, clothed, bathed, and permitted a certain amount of emotional upheaval. We are not expected to assume responsibilities, or to perform difficult duties. Our faults and misdemeanors are not condemned severely, if at all. We are in the position where the world owes us a living; and the world is willing to admit it and behave accordingly. We are, as it were, kings. We can do no wrong; and we do very little else that is difficult or worth while. This is the situation into which we are born, and in which we are reared for a number of years. It is a situation of dependence, and

during the most impressionable and formative years of our lives, we are conditioned to dependence. As we grow up, we are expected to assume more responsibilities, to do more work, to carve out a mode of living for ourselves—in other words, to achieve individual independence. Because the transition from the dependent to the independent state need not be made rapidly, because everyone about us is doing the same thing, because we are taught and helped on the road, because rewards of various sorts are held out to us if we succeed, most of us go along with the crowd and become more or less independent adults. We accept our responsibilities and perform our duties and do not expect too much help from others. We do this because it is the thing to do, and because we must do it; but having reached adulthood and independence, we have not entirely lost the desire for dependence and care to which we were so thoroughly conditioned in childhood. We may not be aware of it, but it is still present, submerged into our unconscious minds, but not eliminated. Who is there who has not occasionally felt that he would like to chuck all this work, or those plans, or these responsibilities, and let someone else worry about them while he relaxed? Practically every one of us has at one time or another, when faced with a difficult situation, chosen the path of least resistance. These are throwbacks to the time when you could do that sort of thing without much censure—when giving in to your whims and inclinations was considered "natural" because you were a child. Those of you who know the unemployment and relief situations realize that there are numbers of individuals who, having been given the opportunity to be cared for by the state, with little or no effort on their part, are exceedingly hesitant to accept jobs which would make them more independent than they possibly could be on relief. Come with me to the Tuberculosis Sanitarium, and I will show you that in the place of the depression and hopelessness that you might expect in the patients as a result of their serious illness, most of them are relatively happy and contented. Why? Because they are in the position where, without pain to make them uncomfortable, they can be completely dependent, with the sanction of society. And finally, and most tragically, consider the individual with schizophrenia. He has come to his adulthood, found life too difficult for him on that plane, and has retreated

to the more immature level where society could make few demands on him. He is a child again.

I have dilated on this point because I consider it highly important. The pull of dependence, though resisted by most of us, is always present; and a good many individuals give way before it—some in one way, some in another. With the tendency to become dependent ever present, the likelihood of giving way before it is apt to depend to a large extent in each individual on his convincing himself that he has a sufficient reason for giving way. Most of us are kept in line by the rules and customs of the civilization in which we live and to which we have been trained to conform. Most of us hesitate to admit to ourselves or to the world that we aren't capable of carrying on; but some of us occasionally succeed in selling ourselves the idea that we have a good reason for quitting and resuming, at least in part, the attractive dependent state of our earlier lives.

I have spent much time on this factor because its implications are of considerable possible importance to individuals with physical defects of all sorts. We see this giving in to the pull of dependence very frequently in individuals who feel that their physical equipment is not up to par, the hard-of-hearing among others. Let me warn you against it—against the use of your defects (and who among us is without them?) as alibis that will seem to excuse your relaxation into defeat in the struggle for a full life. It is a deplorably simple thing to slide into the rut of feeling that not too much should be expected of one—to take the path of least resistance—to feel that one needs help and care—and that one's ability to work and to assume responsibilities and to live in a complete manner is too limited to warrant the effort. Out of this sort of attitude grows hopelessness and depression, whining and begging, complaining and uselessness—and worst of all, a wasting of life and of the worth while things that help to make of living a pleasant and useful activity. It is an admission of defeat—an inability to grow up.

There is one more point that I would stress: In an individual's zeal to overcome an obstacle, to adjust in spite of it, he often exaggerates its importance and the extent to which he must go to conquer it. As a result, he may build his entire life about his defect, permitting a consideration

of it to enter directly or indirectly into his every activity, thus coloring his whole existence with it. Certainly all of us, in mapping out our lives, must keep in mind our strong and weak points—and plan accordingly. To over-emphasize a weakness, to permit it to determine our social contacts and our goings about, to allow sensitivity regarding it to force us to seek only people in situations similar to ours and to avoid rubbing elbows with others—all this, though it may be done in an attempt to adjust most smoothly, is somewhat misdirected and will definitely place a limit on what is to be gotten out of life. Remember that you are not to spend your time *adjusting to deafness*—your job, like mine and like that of everyone else, is to *adjust to life* in its broadest sense. By all means, do everything you can to improve your ability to communicate with people. Learn lip-reading, utilize the available mechanical devices,—these are certainly the first and most important steps to take immediately. Squeeze every drop of help possible from these aids to communication, so that you may attain maximum efficiency, cut down on the emotional tension resulting from constant straining to make out what is being said, and eliminate the feelings of uncertainty that accompany an impairment in the function of any of the senses. It is taken for granted that you will do this, and having done it, you have finished paying tribute to deafness; you have sharpened your tools. Your job, thereafter, becomes the same as that of everyone else. Your psychology is made up, fundamentally, of the same factors as the next man's. Your job is identical with his: the job of measuring yourself, viewing life, and then going ahead in stride to accomplish happiness in the manner most to your liking and according to your abilities.

The psychologic aspects of deafness? Well, I do not know about that, since I am not an ear, but I am a human being, and so are you. You do not live for your ears, you make your ears work for you, and if they fail to produce as efficiently as they should, the work must be done otherwise. What you want is the finished product — and that means adjustment.

With the proper perspective, everyone can adjust completely. There is the real problem—you and everyone would do well not to lose sight of it.

University of Cincinnati.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY.

Meeting of May 19, 1937.

(Continued from June Issue.)

Osteomyelitis of the Cranial Vault (With Lantern Slides). Dr. Joseph E. J. King.

Treatment of osteomyelitis of the cranial vault has been more or less of a hit-and-miss affair until the last few years, so far as operative measures are concerned. The mortality rate was very high. The good results which have been obtained by surgeons who have boldly extirpated the full thickness of the skull well beyond the disease speak for themselves. They are so much better than those previously obtained. This mode of attack on the diseased bone was not advocated until about 23 years ago, and was not seriously carried out until during the last decade. Even now it is not generally appreciated. McKenzie advocated this procedure in his first famous contribution in 1913, and stated that "Once progressive osteomyelitis has set in, the only chance of saving the patient lies in immediate and entire removal of the diseased bone. We can scarcely hope to cure diffuse osteomyelitis by simple drainage, hence limited and partial measures, such as incisions, trephining, etc., are useless." It is remarkable how long it has taken this idea to permeate our own mentalities.

This discussion will consider only the *treatment* of, and surgical principles concerned with, the condition. Etiology, with the pathways of infection, pathology, bacteriology, symptomatology, and diagnosis, will not be dealt with in detail. All, or some, of these phases have been discussed in previous contributions by others. Some of these writers have given detailed and comprehensive review of the literature, while others have stated their own clinical observations and have given reports of cases. Mosher stated that four of the most noteworthy papers which have appeared in recent years on the subject have been those by McKenzie, Bulson, Wilensky, and Furstenberg. I would like to add his name to that list. Since these papers have covered the sub,ect-matter so completely, my purpose in presenting this paper resolves itself into two objectives: first, to emphasize and accentuate the importance of *complete* operation, and second, to give some clinical observations.

CONSIDERATION OF GROSS PATHOLOGY AND SURGICAL PRINCIPLES.

Regardless of whether the initial infection enters a bone of the cranial vault by direct extension from one of the accessory nasal sinuses, the middle ear, or mastoid; by retrograde thrombosis of veins; by thrombophlebitis, retrograde thrombosis and extension through the dura, as suggested by Furstenberg; or by metastasis, the infection is first a local one in the bone, in the majority of cases, and so can be considered from a practical standpoint. Therefore, what could be more logical in combating this otherwise destructive condition than to attack it as early as the diagnosis can be made, and to remove completely the involved area with its source of infection? As was noted by McKenzie, osteomyelitis of the cranial vault very seldom metastasizes to other bones and other organs, in contrast to a similar infection of the long bones. Radical removal should therefore effect a cure. Once it has begun, however, its inevitable tendency is to spread to adjoining portions of the skull and, if untreated, it may run the entire gamut through extradural

and subcutaneous abscesses, cortical and subcortical abscesses, thrombosis of major sinuses, generalized involvement of the skull, meningitis, suppurative leptomeningitis, and sepsis, to death.

It is well recognized by all writers that most cases of osteomyelitis of the frontal bone occur in young people and that the infection tends, for a certain period of time, to localize to one side, or at least be confined to one bone. It is believed that the fibrous tissue which exists between the suture lines before complete closure acts as a barrier to the extension of infection and makes the disease more amenable to eradication.

It is also recognized that the condition frequently begins with stealthy and insidious onset, and that a gross pathologic process, which could and should be removed, exists before the radiographic films show the clear-cut, undeniable, moth-eaten picture of well-established osteomyelitis. F. W. Law and I have reviewed serial X-ray films of cases of osteomyelitis of the frontal bone. We were impressed with the early differences in the appearance of the two sides of the posteroanterior films. These differences are more apparent when the films are reviewed in reverse. If attention is focused on the region of the frontal bone where the eventual breaking down of bone occurs, one can observe an area of less density in earlier films. This is due to softening and partial decalcification of the area. Evidence of the presence of this area is very questionable in the earliest films, or it may not exist at all. After a few days, however, a rounded area of less density is visible at the point where the eventual breaking down will occur. This area is probably more noticeable if viewed from a distance of five or six feet than at a short distance. In some instances in our experience, films were made at the time frontal sinusitis or orbital abscess was present and not again until a definite breaking down of the cranial tables with formation of a subcutaneous or an extradural abscess had occurred. The intervals between the X-ray examinations in many instances were surprisingly long. In view of these observations, we advise that a posteroanterior film be made at intervals of every two or three days with the expectation of finding the area of less density, which gradually appears several days (about ten days to two weeks) before the typical moth-eaten picture makes its appearance. By so doing, the condition may be recognized and operation carried out at an earlier date without so much sacrifice of bone. At least, evidence may be sufficient to warrant an exploratory drill hole at the site.

Although osteomyelitis of the skull may spontaneously occur without any instrumentation by the surgeon, it must be acknowledged that a large number of cases follow instrumentation or operation. This is especially true following any operation upon the sinus. It is believed that fewer cases would develop were the sinus operated upon with purely surgical principles in mind, leaving the question of cosmetic result for a later date. Full, open, and prolonged drainage with iodoform gauze packing after a proper operation would allow the sinus to fill up with firm granulation tissue, and completely obliterate the sinus. Future troubles might be avoided. Mosher holds this view, and Ross Faulkner in a recent conservation gave the same opinion.

DISCUSSION.

Dr. Frederick M. Law: After such a brilliant presentation it seems foolish for an X-ray man to get up and try to say anything, but I do not want you to get the idea from Dr. King's paper that it was easy to find these areas of lessened density. Unfortunately, the X-rays, as far as osteomyelitis is concerned, are rather tardy. They miss the express and take the local, and that gets you in pretty late. If you wait in X-ray interpretation until you get the characteristic moth-eaten appearance, it is just too bad for the patient. If you have films made before any marked change occurs, in other words, if you have a set of stereoscopic films showing no change in the appearance of the frontal bone, but you have symptoms and suspect a change there, do not wait a week or ten days. Do not wait longer than two days,

and preferably 24 hours, to make another set of films, followed by another set in another day or two. The first indication which you see on the film is a little smudge on the frontal bone, about a half-inch to an inch above the superior margin of the frontal sinus. That smudge resembles a mark made on the clear white frontal bone as if made with a dirty finger. It is undetectable unless you have previous films which do not show it and unless you make careful observations. Place the film in an illuminating box and stand five or six feet away from the film. It is surprising how it becomes visible as you get farther away. Dr. King and I sometimes stand and look ten or 15 minutes at those films without saying a word and then we see the area. It is the only early sign you can get. It is an absolutely certain sign, but you must educate your eyes to see it. I urge you, in all the early suspected cases, not to hurry. Look at the films. Go back an hour later and look at them again. Eventually you will train your eyes to the point where, as Dr. King says, you can detect the faint change in density.

Infection of the Carotid Canal Area Cured by Cervical Operation.
Dr. Mervin C. Myerson.

While it is true that this patient had an infection of his carotid canal and we drained it and cured the carotid canal infection, he had other things which eventually caused his death. However, I would like to interest you in the infections of the carotid canal area in petrous pyramid suppuration, its significance and how it can be attacked in some cases in a fairly simple and conservative manner.

(Diagram.) If this is the right temporal bone, looking at it from before backward, we know that the internal carotid artery comes up the lateral wall of the neck, very close to the posterior wall, and just beyond the tympanic plate it enters the vertical portion of the carotid canal and then makes a turn and goes horizontally a distance, and then goes up into the cavernous sinus area. The average length of the inferior plate of bone would be about a half-inch for the carotid canal. In our studies of petrous pyramid suppuration we have come to appreciate that there are two groups of infections from the standpoint of their location. The infection localizes in cellular bone either above or below in the petrous pyramid; in other words, the so-called infra- and supralabyrinthine or infra- and supracochlear areas, or any other terminology you wish to use. When a tract of cells in the inferior portion of the pyramid in relation to the carotid-cochlear angle area, or a tract of cells that would have its beginning just below the posterior semicircular canal, either or both of these groups of cells becomes infected, we have a localization in the posterior portion of the petrous pyramid. When that infection gives way to the production of pus, we have a collection of pus usually in the inferior portion of the apex cavity. When the pus localizes in the carotid canal, there should be, as Stacy Guild stated on purely theoretical evidence, a Horner's syndrome. The case I am about to report developed a Horner's syndrome. Another very important and significant symptom which, so far as I know has not been mentioned in this disease, is pain deep in the glenoid fossa on movement of the temporomandibular joint. We have two signs and symptoms which are not generally known and which have not been observed heretofore, as far as I know. It stands to reason, if we can go through the neck to the roof of the retropharyngeal space, and if there is pus, we will be able to drain pus from the inferior aspect of the petrous. It is possible, of course, either for pus in the apex cavity to erode through the posterior wall of the carotid canal or upward into the apex. Our experience relates to a man, age 44 years, who was first seen by us when we took over the service in February of this year. He had been admitted for surgery of the sinuses.